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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontology; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Membership in the Academy is by invitation only, but non-members are welcome to attend any of the Academy meetings.

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JOHN OPPIE McCALL, A.B., D.D.S.
Jericho, New York

Dr. John Oppie McCall has long been active in the promotion of the highest of ideals for dentistry and particularly periodontology. He is well-known for his active role in teaching and writing, which dealt with many aspects of dental science.

Dr. McCall interrupted his active program of teaching and dental practice to become director of the Guggenheim Dental Clinic, a position which he held until his retirement in 1948. He returned then to private practice of periodontology, and retired from practice late in 1960.

In 1911, Dr. McCall devised an excellent set of scalers known as the "McCall Scalers" which have been widely used over the years. He is the author of a textbook on clinical periodontology and of five other dental textbooks, as well as innumerable shorter articles.

Dr. McCall has served organized dentistry as editor of the First District Dental Society and the New York Journal of Dentistry, and has served on many committees. Dr. McCall and his lovely wife have four children, all but one of whom have continued the medical trend. He and his wife now reside at 262 Fairhaven Mall, Jericho, New York.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED SIXTY ONE

The Cementum: Its Role In Periodontal Health and Disease*

by DONALD A. KERR, D.D.S., M.S.,** *Ann Arbor, Michigan*

THE cementum is a specialized calcified tissue of mesenchymal origin which provides for the attachment of the periodontal fibers to the surface of the root. It consists of 45 to 50 per cent inorganic material and 50 to 55 per cent organic material with the inorganic material in a hydroxyl apatite structure.

The primary cementum is formed initially by appositional growth from the dental sac and later from the periodontal membrane under the influence of cementoblasts. It is formed in laminated layers with the incorporation of Sharpey's fibers into a fibrillar matrix which undergoes calcification. Cementum deposition is a continuous process throughout life with new cementum being deposited over the old cemental surface. Cementum is formed by the organization of collagen fibrils which are cemented together by a matrix produced by the polymerization of mucopolysaccharides. This material is designated as cementoid and becomes mature cementum upon calcification. The significance of the continuous deposition of cementum has received various interpretations.

1. Continuous deposition of cementum is necessary for the reattachment of periodontal fibers which have been destroyed or which require reorientation due to change in position of teeth. It is logical that there should be a continuous deposition of cementum because it is doubtful that the initial fibers are retained throughout the life of the tooth, and therefore new fibers must be continually formed and attached by new cementum. There is without question some wear and tear damage due to traumatism which would necessitate replacement. However, some question the need of new cementum for reattachment of fibers associated with functional reorientation. If we accept the premise that the fibers of the periodontal membrane are not continuous from bone to cementum but that cemental fibers and alveolar fibers are united by an intermediate layer, then functional reorientation can occur in the intermediate layer and reattachment by new deposition of cementum is unnecessary. The report of Gustafson and

*Read before the Annual Meeting of the American Academy of Periodontology, October 12-15, 1960, in Santa Monica, California.

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Fig. 1. Resorption of cementum, dentin, and bone in an area of trauma. The resorption is inactive and the widened periodontal space is filled with fibrous tissue. Note active osteogenesis on outer surface of alveolar process.

Fig. 2. Area of resorption of cementum and dentin repaired by cellular and acellular cementum. This is the root of a deciduous tooth. The enamel organ of the succedaneous tooth is evident at the right.

Persson[4] demonstrated that change in direction of Sharpey's fibers in cementum and bone has a relationship to the deposition of both cementum and bone. This suggests that new layers of cementum become necessary when change in position produces bending of Sharpey's fibers at the point of insertion into cementum.

2. Various authors have suggested that after deposition, cementum underwent aging and became nonvital. When the surface cementum became nonvital, the attachment to the tooth had to be re-established by apposition of a new layer of vital cementum.

3. It also has been indicated that cementum apposition is dependent upon tooth movement to provide the necessary space for apposition of new layers. This is in part true, but cementum deposition can be stimulated by hyperfunction associated with the widening of the periodontal space, by resorption of alveolar bone in response to occlusal traumatism, and cementum deposition may also be induced during the reparative phase of periodontal injury or tooth fracture.

4. The continuous deposition of cementum may be considered as an example of a normal biologic characteristic of all forms of calcified tissue. When tissue is fully calcified, it is susceptible to resorption, while uncalcified matrix is resistant to resorption. In intrachondrial bone formation the portion preformed by cartilage does not undergo resorption or replacement until calcification has occurred. After the cartilage has calcified, it is then resorbed by osteoclastic action. Bone, cementum, and dentin are all protected at the point of contact with connective tissue by a layer of uncalcified matrix. With destruction of the formative tissue uncalcified matrix is not present and the calcified tissue is resorbed by osteoclastic action in the zone of repair. Resorption of all calcified tissue can be produced by osteoclastic action. It is logical to postulate that the continuous deposition of

Fig. 3. Epithelial attachment in area of resorption at the bottom of a periodontal pocket. The space between epithelium and cementum is due to shrinkage in preparation.

Fig. 4. Small area of cemental resorption filled with proliferating epithelial rest. Larger nests of epithelium in the periodontal membrane. The bottom of the epithelial attachment at the base of the pocket is evident in the upper right corner.

cementum is a protective biological process without which the supporting structure could not be maintained. This is supported by Zander and Hürzeler's[10] study in which they observed a straight line relationship between the thickness of cementum and age. This would suggest that continuous deposition of cementum was a biologic characteristic of calcified tissue which of necessity had to occur at a constant rate to provide the necessary protection of the mature calcified tissue. The cementum is more resistant to resorptive action than bone because of the nature of its vascular supply. When pressure is increased in the periodontal space, there is an increased intravascular pressure within the periodontal tissue which is transmitted by the vessels contained within the periodontium to the marrow and haversian spaces of the adjacent bone. The altered vascular supply effects the endosteum and stimulates bone resorption. Bone changes cannot be interpolated into cemental changes because all bone is vital and influenced by circulation, while only the surface of the cementum can be affected by changes in the circulation of the periodontal membrane. The only positive statement which we can make relative to the resorption of the two substances is that cementum is more resistant to resorption than bone even though it is produced in both tissues by osteoclastic action.

Cementum which is deposited after the initial layer covers the root may be of two varieties: it may be acellular like the initial cementum, or it may be cellular in character. The cellular cementum resembles bone in that formative cells are incorporated in lacunae spaces from which there are radiating canaliculi. Because of morphological similarity to bone, it has been designated as osteocementum. The lamina of osteocementum are thick and bulky in comparison to the lamina of primary cementum. The lamina contain a variable number of lacunae oriented parallel to the surface and with anastomosis of canaliculi. This provides a limited circulation to maintain the vitality of a thicker lamina. Cellular cementum appears to be deposited in broad

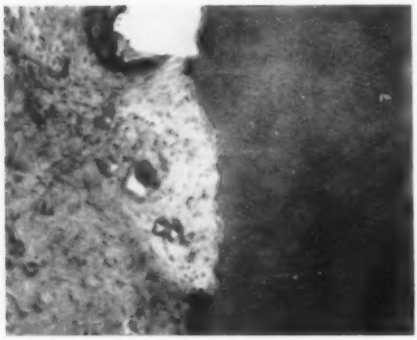

Fig. 5. Area of resorption at the bottom of the pocket just below the level of the epithelial attachment. No evidence of repair and very limited inflammation.

lamina when there is a rapid demand for a large amount of cementum to compensate for extensive movement or repair. Held[5] states that cellular cementum is produced by the incorporation of fibroblasts in the matrix at the time of formation and produces the cementocytes, while cementoblasts are not incorporated into the matrix of primary cementum. Thus the formation of the two types of cementum involve different degrees of histodifferentiation. This is analogous to the formation of fibrillar and mature bone. Cellular cementum can be covered by acellular cementum.

Investigations by Stones[9] indicate that primary (acellular) cementum is impervious while cellular cementum is porous. When cellular cementum is in contact with dentin, the cementocytes at the cementodentinal junction remain vital, while those slightly removed from the junction become devital with subsequent apposition of new lamina. This observation may carry some significance when we consider the cementum in disease.

Cemental hyperplasia occurs in response to heavy functional demand. When individual teeth or small groups of teeth are placed in heavy function, the surface of the cementum becomes irregular with spurs extending into the areas of principal fiber attachment. This undulating pattern increases the area of periodontal attachment and strengthens the supporting mechanism (Fig. 7). In heavy function the cementum is considerably thickened in the apical portion of the root. These changes indicate that cementum deposition is influenced by function as well as by movement of the teeth.

Increased deposition of cementum also occurs in association with periodontal inflammation, and changes which stimulate reparative proliferation may be manifest as hypercementosis.

Certain alterations in cementum have been suggested or postulated to be of importance in periodontal disease, and certain observations have been made which perhaps are worthy of note although their full significance cannot be explained.

Some authors, Gottlieb[1,2] in particular, have suggested that periodontal disease was a disease process of cementum, so-called cementopathia. It is difficult to see how this could be true when cementum is basically a nonvital calcified material which has to be continually "vitalized" by apposition

Fig. 6. Areas of resorption of cementum exposed in a periodontal pocket. The exposed cemental surface is covered by calculus.

of new cementum. Cementum is a product of mesenchymal tissue, and if there is a dysplastic process present, it must be one of the mesenchymal formative tissue. Such processes are rarely demonstrated, but a few examples have been observed in children in which there was spontaneous shedding of deciduous teeth, especially incisors, without attendant root resorption. Microscopic sections of such teeth demonstrate a complete absence or a very limited quantity of cementum on the entire root surface. This appears to be a dysplastic process of mesenchymal origin. All of the cases I have studied involved the deciduous dentition except one which involved the permanent teeth of a boy aged ten.

Hürzeler and Zander[6] studied cementum apposition in periodontally involved teeth and concluded that cementum apposition was less in periodontally involved teeth than in normal teeth. It appeared that cemental apposition decreased with advancing age in periodontally involved teeth. According to the authors this would support the contention that periodontal disease was a disease of the attachment apparatus, and as a result of the process cementum apposition was reduced. However, it does not indicate that the process is a dysplasia of the cementum.

Fig. 8. Enamel pearl in the bifurcation which is covered by a nodule of hyperplastic cementum.

Fig. 7. This shows an area of cemental hyperplasia which has now been exposed by the development of a periodontal pocket. This provides a rough cemental surface for the accumulation of debris and the firm attachment of calculus similar to the pattern produced by resorption.

One would anticipate that periodontally involved teeth would have less cementum because of the detachment of periodontal membrane associated with pocket formation. Also, because these teeth were mobile, attrition with associated continuous eruption and apical thickening of cementum did not occur. This does not indicate that periodontal disease is associated with decreased cementum deposition.

Several writers have suggested that the cementum and dentin of the cervical area exposed in a periodontal pocket was softer than unexposed cementum and that it also was more porous than unexposed cementum. The cementum at the cervical area is acellular which Stones[8] has demonstrated

to be nonporous. However, exposed cementum may become decalcified after exposure and would then be soft and porous and an unsatisfactory environment for reattachment of periodontal fibers.

Rautiola[7] studied the hardness of cementum in both normal and periodontally involved teeth. He found that the cementum exposed by periodontal disease was of the same hardness as unexposed cementum of the same tooth or of normal teeth. This would indicate that the clinical impression expressed by Riffle[8] and others was incorrect.

In studying the root surfaces of periodontally involved teeth, areas of cemental resorption are frequently observed and their character and location suggest a varied etiology. One type of cemental resorption is seen in association with traumatism of a severity to produce compression of the periodontal membrane to the point of necrosis. As a part of the reparative process, cemental resorption is produced (Fig. 1). Such areas of resorption may be partially or totally repaired by new cementum deposition (Fig. 2). If apical migration of the epithelial attachment reaches such a level of cemental resorption, it appears to establish the same attachment as is provided on primary cementum (Fig. 3). In some areas of resorption due to traumatism there is proliferation of epithelial debris as a part of the reparative process and the epithelium attaches to the area of resorption (Fig. 4). These changes are identical to those described by Gottlieb[3] as typical of periodontosis.

Another form of cemental resorption observed in periodontitis is seen at the level of active inflammatory response in the bottom of the pocket, just apical to the epithelial attachment. In this zone principal fibers are partially destroyed or detached, there is active inflammation and fibroblastic proliferation, and cemental resorption. The resorption is usually shallow and limited in area. There is no evidence of repair in such areas, and they may be surfaced either by mesenchymal or epithelial tissue (Fig. 5).

A third type of resorption is seen in the cementum of the root exposed in a periodontal pocket. It is shallow and often appears to have been multicentric in origin. It does not have the same pattern as described for the other two types. The impression obtained from studying these areas is that of resorption of devital cementum by mesenchymal elements of the granulation tissue replacing the epithelial lining of the pocket. However, it is impossible to determine whether this might not have been initiated prior to exposure of the cementum into the pocket. Such areas are conducive to the accumulation of debris and the deposition of calculus (Fig. 6). Deposition of calculus into the irregular areas of resorption provides a firm attachment and makes complete removal extremely difficult and may explain some failures of reattachment and/or pocket elimination (Fig. 7).

Localized areas devoid of cementum, not to be confused with resorption, are present due to the persistence of Hertwig's epithelial sheath in selected areas. When the epithelium persists on the surface of the root, the dental sac or periodontal membrane is not influenced to produce cementum. Such areas of agenesis may ultimately be involved in pocket formation and become exposed into the pocket to be confused with areas of resorption.

From these observations it is suggested that cemental resorption may be a manifestation of periodontal disease before, during, and after pocket formation.

Anomalies of cementum deposition may also be associated with enamel pearls either in the form of aplasia of cementum or hyperplasia with irregular nodular masses over the pearl. Because of the frequency with which they occur in the bifurcation areas, they may influence the development or progress of the pocket into the bifurcation (Fig. 8).

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NEW DIPLOMATES OF THE AMERICAN BOARD OF PERIODONTOLOGY

1961

The American Board of Periodontology announces that the following dentists were certified as diplomates at their last meeting:

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Studies on Experimental Calculus Formation in the Rat*
I. Effect of Age, Sex, Strain, High Carbohydrate, High Protein Diets

by PAUL N. BAER, ROBERT M. STEPHAN AND CARL L. WHITE,** *Bethesda, Md.*

DENTAL calculus or calculus-like deposits have been observed in experimental animals by several investigators.[1-8] During the past ten years one of us (RMS) repeatedly found calculus or calculus-like deposits on the molar teeth of rats fed certain fine particle diets which were used in nutritional experiments and experiments on the production of smooth surface caries. It was also observed that calculus rarely formed on the teeth of rats which had been fed purina laboratory chow in the usual form of hard pellets. The calculus formation did not appear related to the development of periodontal disease which also was found to have occurred in some of the animals. In these caries studies, in which calculus was observed as an incidental finding, the occurrence of calculus was most uniform in rats which had been fed Diet 580 (66% sucrose, 32% skim milk powder, and 2% whole liver substance). It was also observed that of the different strains of rats available from the N.I.H. animal production section, the Holtzmann-Rolfmeyer strain of rats produced calculus most uniformly. These observations have furnished a basis for studies on experimental calculus formation in the rat. Thus, Stewart and Burnett[1] have recently published a study on calculus formation in the Holtzmann-Rolfmeyer rats using Diet 580 as a starting point. The present study was initiated similarly, and was designed to evaluate methods for scoring calculus and to further explore the effects on calculus formation of various experimental factors, including the effects of sex difference, strain difference, and differences in the types of carbohydrates and the amount of protein in the diet.

Any hard adherent deposit which had been formed on the tooth surfaces exposed in the oral cavity was considered to be calculus.

METHODS AND RESULTS

A. Comparison of methods of scoring: The animals were received from the animal production unit N.I.H. at weaning, and were placed in raised bottom wire mesh cages and fed diet and water ad libitum. Table I gives the composition of diets. At the end of the various experimental periods, the animals were sacrificed with chloroform. The heads were removed, defleshed, and the molar teeth on one half the jaw of each arch were examined microscopically at 10X magnification and scored for the presence of calculus.

Two methods for scoring calculus were evaluated. One was the following system which had been used for the past 10 years in scoring calculus in rats on caries experiments by one of us (R.M.S.).

0—no calculus present.

1—a very small amount of calculus present usually limited to the mesial surface of the first molar, Fig. 1.

2—a small amount of calculus present usually limited to the mesial, buccal and lingual surface(s) of the first molar.

3—an intermediate amount of calculus present usually located on both the 1st and 2nd molar teeth, Fig. 2.

4—a large amount of calculus present usually present on all three molar teeth, Fig. 3.

Since calculus deposits were more or less symmetrical for either side, each arch received but one number.

In the second technique each tooth was scored separately, as follows:

Each molar tooth received one point for each exposed tooth surface possessing calculus. In addition, if calculus was heavy on any or all surfaces of

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*Presented at the meeting of the American Academy of Periodontology, Santa Monica, California, October 1960.

**National Institute of Dental Research, National Institutes of Health, U.S. Public Health Service, Department of Health, Education, and Welfare, Bethesda 14, Maryland.

TABLE I
Composition of Diets

High Sucrose (Diet 580)		*High Glucose*		*High Cornstarch*	
Skim milk	32%	Skim milk	32%	Skim milk	32%
Liver Powder*	2%	Liver Powder	2%	Liver Powder	2%
Sucrose	66%	Glucose	66%	Cornstarch	66%

High Protein (111)		*Control Diet (normal diet) 112*	
Casein	62%	Casein	25%
Crisco	8%	Crisco	8%
Sucrose	13%	Sucrose	50%
Salts (HMW)	4%	Salts (HMW)	4%
Liver Powder*	5%	Liver Powder	5%
Brewers Yeast	3%	Brewers Yeast	3%
"Alphacel"	5%	"Alphacel"	5%

Equi-Caloric Diets

	Diet 4		*Diet 7*	
Ingredients	%	*Calories/100 gm.*	%	*Calories/100 gm.*
"Alphacel"	0	0	22	0
Sucrose	66	264	26	104
Casein	18	72	18	72
Crisco	5	45	23	205
Brewers Yeast	3	8	3	8
Liver Powder*	5	15	5	15
Salts (HMW)	3	0	3	0

*Desiccated, N.F. (Wilson)

a given tooth, another point was added. Thus, the maximum score for each molar tooth was:

1st molar 4 (it has 3 exposed surfaces)
2nd molar 3 (it has 2 exposed surfaces)
3rd molar 4 (it has 3 exposed surfaces)

The values of the individual teeth were then added to give the total score.

When the two methods were evaluated, it was found that the same conclusions were obtained. Therefore as the former system was faster and easier, it became the method of choice.

B. Comparison of calculus formation in males and females: Twenty male and twenty female weanling rats of the Sprague-Dawley strain were maintained in individual wire cages and fed a diet containing 66% sucrose (diet 580, Table I) for a period of 70 days. Another group of 6 males and 6 females were sacrificed after 30 days on the experimental diet. At the end of the various test periods the molar teeth were scored for calculus.

Results: It was evident from Table II, that the average scores were very similar for male and female whether the maxillary or mandibular teeth were used for comparison.

C. Effect of strain: Seventy-two rats, six of each sex of strains M520, Osborne-Mendel, Holtzmann-Rolfmeyer, N.I.H. Black rat, Fischer, and Sprague-Dawley were fed the same diet as in the previous experiment for a period of 70 days.

All six strains formed calculus with the average maxillary scores between 2.3 for strain M520 and 3.5 for the Fischer and Black rat strains. The average mandibular scores ranged between 1.6 for strain M520 and 3.6 for strain Black rat. The importance of these findings is that all six strains were capable of forming calculus on this particular diet.

D. Effect of age at onset of experimental period: Seven groups of Sprague-Dawley rats were 1, 2, 3, 4, 5, 6 and 11 months

Fig. 1. Note calculus on mesial surface of first molar only, for a score of 1.

of age respectively at the onset of the experiment period. All other experimental procedures were the same as those used in part C, above.

Table III lists the results from this experiment. It is evident that, if one compares the animals which were 1, 2, or 3 months of age with those 6 to 11 months of age at the onset of the experiment, that the older rats did not form caluclus as readily as the younger ones. Statistical tests reveal significant differences, ($p=0.01$) between these age groups for both maxillary and mandibular scores. By repeating this experiment, and comparing just the 1 month with the 11 month old animals, the previous findings were confirmed.

E. Effect of different carbohydrates: In our previous experiments sucrose was the only carbohydrate used. The question was raised as to whether the deposition of calculus might be influenced by the nature of the carbohydrate in the diet, i.e., whether it was a monosaccharide, a disaccharide, or a polysaccharide. Thirty-six Sprague-Dawley rats of both sexes were divided into 3 groups of 12 animals per group. One group was fed a diet high in sucrose, (Diet 580) one diet high in glucose, and one a diet high in cornstarch, (Table I). At the end of 30 days, the animals were sacrificed. (See experiment A in Table IV).

Results: When the average scores per animal are compared, it can be seen that the diet containing the cornstarch produced the greater amount of calculus. The experiment was repeated (Experiment B, Table

Fig. 2. Calculus is present on both the first and second molars for a score of 3.

Fig. 3. Calculus is present on all three molar teeth for a score of 4.

IV) but only sucrose and cornstarch diets were compared. The results were similar to those in part A. The difference between these two diets were statistically significant at the $p = 0.01$ level.

F. Caloric effect: To determine whether the effects noted in the above experiments were due to the carbohydrate per se, or were due to a caloric effect, two diets of identical caloric content were tested. One, Diet #4, was high in sucrose and low in fat, while Diet #7 was high in fat and low in sucrose, (Table I). Each group contained 27 animals and the experimental period was 30 days.

Results: There was no difference in the weight gained by these animals on these two diets. Table V shows that the two diets, of equal caloric content, and somewhat similar physical consistencies, produce almost identical amounts of calculus deposits.

G. Effect of a high protein low carbohydrate diet: A semi-purified diet (111) containing 62% protein in the form of casein was compared to a Purina Laboratory Chow diet, which contains 25% protein, and to a semi-purified diet (112) which also contains 25% protein, Table I. The animals used were Sprague-Dawley weanling males, and the experimental period was 30 days.

Results: There was no difference between the high protein-low carbohydrate diet and the semi-purified control diet. The animals on the Purina diet, however, did not form calculus, Table VI.

DISCUSSION

Lack of sufficient friction from food during mastication has been held responsi-

TABLE II
Comparison of Calculus Formation in Males and Females

Diet	*N*	*Duration of Treatment*	*Maxillary Score**		*Mandibular Score**	
			Male	*Female*	*Male*	*Female*
580 (66% sucrose)	40	70 days	3.1 ± .19	3.2 ± .20	2.6 ± .37	2.7 ± .37
	12	30 days	2.0 ± .10	2.8 ± .18	.9 ± .16	1.4 ± .39

*Mean scores and standard errors

TABLE III
Effect of Age at Onset of Experimental Period

Age (in Months)	*N*	*Maxillary* Score*	*Mandibular* Score*	*Average Score* per Animal*
1	21	2.9 ± .17	2.0 ± .32	4.9 ± .43
2	6	3.3 ± .33	1.2 ± .40	4.5 ± .62
3	6	2.8 ± .33	1.7 ± .55	4.5 ± .81
4	6	2.2 ± .12	1.5 ± .43	3.7 ± .49
5	6	2.0 ± .36	0.7 ± .42	2.7 ± .76
6	6	1.5 ± .34	0 ...	1.5 ± .34
11	18	0.8 ± .10	0 ...	0.8 ± .09

*Mean scores and standard errors

TABLE IV
Effect of Different Carbohydrates

Diet	*N*	*Mean Calculus Scores*		
		Maxillary	*Mandibular*	*Whole Mouth**
			Experiment A	
Sucrose	11	2.5	1.5	4.1 ± .41
Glucose	12	3.2	1.5	4.7 ± .45
Cornstarch	12	3.4	3.1	6.5 ± .29
			Experiment B	
Sucrose	12	2.7	1.1	3.8 ± .33
Cornstarch	12	3.4	2.8	6.2 ± .68

*Mean scores and the standard errors

TABLE V
Effect of Diets of Equal Caloric Content

Diet	*N*	*Maxillary* Score*	*Mandibular* Score*	*Mean Score* per Animal*
#4 (high sucrose, low fat)	27	2.4 ± .13	1.1 ± .20	3.5 ± .32
#7 (high fat, low sucrose)	27	2.7 ± .20	1.3 ± .25	4.1 ± .41

*Mean scores and standard errors

TABLE VI
Effect of a High Protein Diet

Diet	*N*	*Average Maxillary Score*	*Average Mandibular Score*
high protein	15	1.7	0.1
semi-purified control	14	1.6	0.1
Purina Lab. chow	15	0	0

ble for calculus formation in such animals as the ferret[5,6,7] and the dog.[8] In the golden hamster, on the other hand, no explanation was offered to explain the heavy deposits of calculus which formed when the animals were fed diets containing large amounts of wholemeal flour, wheatmeal bread or white maize.[5]

The effects we observed with the different carbohydrates might have been due partly to the physical consistency of the diet. The cornstarch diet had the finest physical consistency and produced the greatest amount of calculus. The fact that it was also a polysaccharide may also have played a role.

The results reported here on the effect of a high protein diet on calculus formation are somewhat different from those reported by Stewart and Burnett. Our results indicated that a diet containing 62% protein, and 13% sucrose when compared to a diet containing 25% casein and 50% sucrose and identical in all other ingredients, did not produce a significant difference in calculus formation. These two diets appeared to have about the same physical consistencies. It is possible that the results obtained by Stewart and Burnett might have been partly due to the physical consistency of their protein diet rather than to its protein content per se. This premise appears reasonable when we note that the Purina Laboratory Chow, which contained 25% protein did not produce calculus. On the other hand, the semi-purified diet which contained 25% protein, and was of a powdery consistency, did produce calculus.

Another interesting difference between these findings and those reported by Stewart and Burnett is the fact that the calculus in their animals was limited to the teeth in the maxillary arch, while in our animals calculus was found on the molar teeth of both arches, and in a much shorter period of time. These differences could also have been a result of the different strains of rats employed.

The formation of calculus in all six strains of animals on the experimental diet is highly suggestive that the effect produced is one that might apply to all strains of the rat family, and possibly to other species. This latter phase of the problem is under investigation in mice and hamsters.

SUMMARY AND CONCLUSIONS

1. Dental calculus can be produced in Sprague-Dawley weanling rats in 30 days on diets of a fine physical consistency and relatively high caloric content.

2. Older rats (6-11 months of age) do not form dental calculus as readily as younger animals (1-3 months of age).

3. A carbohydrate diet containing 66% cornstarch produced more calculus than similar diets containing either 66% sucrose or 66% glucose, or a diet of equal caloric value in which fat was substituted for the carbohydrate.

4. All six strains of rats tested were capable of forming calculus on the molar teeth of both jaws on a high carbohydrate diet. More calculus is formed on the maxillary than the mandibular teeth.

5. A diet containing 62% protein and 13% sucrose did not produce any more calculus than one containing 25% protein and 50% sucrose, all other ingredients being identical.

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Orabase and Orahesive: Toxicity Studies in the Rat

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TWO new preparations designed specifically to adhere to the oral mucous membranes have been introduced recently to the profession.[1] The first of these, a paste* preparation containing gelatin, pectin, and carboxymethylcellulose in a mineral oil-polyethylene base, has been found useful as a vehicle for the application of drugs to oral mucosal lesions and as a topical protectant for allaying the discomfort of various acute and chronic oral lesions. With slight modification of the formula as to flavor, this same preparation has been successfully used as a denture adhesive. The above ingredients, without the addition of the mineral oil-polyethylene base (the second preparation) have been used as a powder** for similar purposes.

Inasmuch as either preparation would or could be applied over prolonged periods of time, the question of local or systemic toxic potential must necessarily be considered. Thus, it was concluded that chronic toxicity studies of these two preparations would be desirable.

METHODS

Thirty 200 gram male Sherman rats were used as the basis for this toxicity study. Group I was composed of 10 animals who received Orahesive *paste* mixed in their diet (0.4 gram per 15 grams of food) for a period of twelve weeks. Owing to the proportionately very high mineral oil content of the ingested food (which might interfere with Vitamin A absorption), five* of these animals received weekly gluteal muscle injections of 75 units of Vitamin A; the remaining five animals did not receive Vitamin A.

Group II was composed of 10 animals into whose mouths 100 mg. of the *powder* preparation was insufflated twice a day, five days a week, and continued for twelve

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The adhesive preparations—Orabase* and Orahesive** powder—were supplied by The Squibb Institute for Medical Research.

*One of the Vitamin A treated animals in Group I expired at the end of the fourth week in the experiment. The lungs were observed to be highly congested but the carcass was too decomposed to permit useful further study.

weeks. It was not uncommon for the tongue and oral tissues of the rat to remain in a somewhat "glued" state for periods of an hour or more following application of this material. Eventually, salivation, drinking, and ingestion freed these tissues of the applied material.

Group III was composed of 10 untreated animals serving as controls. Five of these rats received injections of 75 units of Vitamin A in the gluteal muscle once a week and five did not.

All animals were individually caged and were fed "Old Formula" Purina Laboratory chow ad libitum. The animals were observed over an experimental period of twelve weeks and were sacrificed at the end of this period.

The animals' heads were placed in 10% formalin and fixed for at least two weeks. Tissue specimens were obtained from the buccal mucosae, tongue, and lips of each animal for histologic study. All tissue specimens were stained with hematoxylin and eosin and remained coded ("blind") throughout the period of histologic study.

RESULTS

Clinical observations for evidences of oral mucosal toxicity were entirely negative for all animals. Histologic studies of the tissue specimens revealed no significant changes in the treated groups (Groups I and II) when compared with the control group (Group III).

DISCUSSION

Although clinical and histologic abnormalities of the oral soft tissues resulting from use of the adhesives were not detected, these findings cannot be construed as being indicative of negative findings for the rest of the body. However, autopsy tissue specimens of various internal organs were also taken for histologic study. The results of these studies, also essentially negative as to untoward findings, have been reported separately.

Care must nevertheless be taken in the projection of these data (in rats) to the human species, since differences in terms of dose response in particular and individual tissue responses in general are often of great importance.

SUMMARY

Controlled chronic toxicity studies in rats on two new adhesive preparations, Orabase and Orahesive powder, designed specifically to adhere to the oral mucous membranes, were carried out over a twelve-week period of observation.

Clinical and histologic studies of the soft tissues of the mouth did not reveal significant changes in the treated rats when compared to those of the untreated control animals.

The authors express their appreciation to Eric Beube, Mrs. Tanya Genningsen, Mrs. Aldonna Skislak, and Mrs. Helene W. Kutscher.

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The Albert Einstein College of Medicine, Department of Dentistry will sponsor a five day participation course in Clinical Periodontics, Monday to Friday, October 30, 31, November 1, 2, 3, 1961. For further information please write Director, Postgraduate Dental Program, Albert Einstein College of Medicine, Eastchester Road, Morris Park Avenue, Bronx 61, N.Y.

The Occurrence of Bacteria within the Clinically Healthy Gingival Crevice

by J. B. GAVIN AND A. A. COLLINS,* *Dunedin, New Zealand*

IT appears to be generally agreed that, in gingival crevices containing calculus or deposits, bacteria exist in substantial numbers. However, the presence or absence of bacteria within the healthy gingival crevice is the subject of considerable controversy.

In 1937 Hatton[1] thought it unlikely that conditions within the gingival crevice were favourable for bacterial growth. Later Waerhaug and Steen, (1952)[2,3,4] devised a method for the bacteriological investigation of the gingival crevice and came to the conclusion that the gingival crevice free from calculus or deposits is, as a rule, sterile. Brill, (1959)[5] has also demonstrated the removal from the crevice of introduced particulate matter including bacterial cultures.

This view that the healthy gingival crevice is sterile is not universally held, many authorities believing otherwise. The constant presence of leukocytes and plasma cells in the connective tissues about the gingival crevice, is to Orban[6] an indication of the presence of bacteria within the crevice. Other authors[7,8] support the view that bacteria are present within the crevice and state that even in the healthiest mouths the gingival crevice contains a myriad of organisms of many different types. Boyd and Rosenthal, (1958)[9] appear to be the only investigators to have bacteriological evidence supporting this view. They found bacteria in 95.9% of adult healthy gingival crevices.

It is interesting to note that the authorities mentioned above also disagree as to the apical extent of the gingival crevice. In this study the gingival crevice was taken as that area between the free gingiva and the tooth surface and it was entered only until resistance was felt. This was thought to correspond with the most coronal portion of the "epithelial attachment" or slightly beyond.

Due to the wide divergence of opinion it was decided to study further, by an independent method, the bacteriological status of the clinically healthy human gingival crevice.

METHOD AND MATERIAL

To overcome the problem of contamination from the tooth and gingival surfaces outside, but adjacent to the crevice, the following method was devised: Using a saliva ejector and cotton rolls placed lingually and in the oral vestibule the selected area was isolated. The gingival and tooth surfaces were carefully dried with gauze, care being taken to avoid introduction to the crevice of any foreign material. A 5mm length of 19 gauge hypodermic needle with a smoothed 1mm bevel was held in a fine pair of artery forceps and sterilised in alcohol and a spirit flame. Once cool, this tube was placed perpendicularly to the tooth surface, and with the bevel facing occlusally, on the labial or buccal free gingival margin (Fig. 1). By carefully rotating this tube occlusally until it made an angle of approximately twenty degrees with the tooth surface the gingival margin could be displaced laterally from the tooth (Fig. 2). Care was taken to ensure that the tube was not "pushed into" the crevice, the action being one of rotation, and that contact with tooth or gingiva was made with the external surface of the tube only.

A sterile paper point passed through the tube in this position will collect material from the depths of the gingival crevice. The points, held in sterile tweezers, were passed gently into the crevice until resistance was felt. The points entered the

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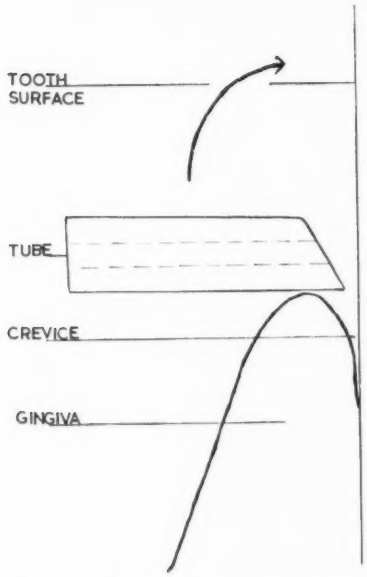

Fig. 1. Diagram showing tube placed on free gingival margin.

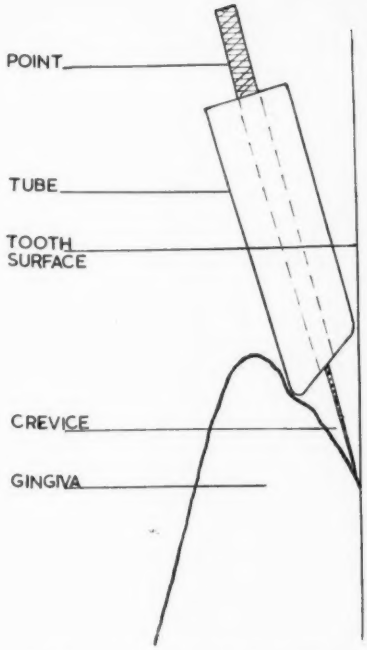

Fig. 2. Diagram showing tube in position with paper point passed into the gingival crevice.

crevices to a depth of 1-2mm usually, corresponding, we believe, to the level of the coronal end of the epithelial attachment or slightly beyond. At no time did the paper points touch either tooth or gingival surfaces external to the crevice.

As a control of the method to ensure that the material was collected from the depth of the crevice and was not contaminated by the tube, the tube was placed as described, removed, and a sterile point passed through the tube.

The paper points with the absorbed material were cultured at 37°C in Difco dextrose broth for three days for aerobic organisms and in Robertson's meat broth for seven days for anaerobic organisms. Those cultures showing no evidence of growth after these times were considered to be negative cultures.

Male dental students who maintained a high standard of oral hygiene were used in this study and the crevices about incisor, canine, and premolar teeth, with approximately equal numbers from each jaw, were investigated. No crevices were considered if any signs of deposits on the teeth or inflammatory changes involving the gingivae were clinicaly evident. Each culture was made from a different gingival crevice.

This method was used to investigate the gingival crevices of twenty-six subjects. In a second group of seven subjects the method was used following the application for three minutes of an iodine glycerol mixture (equal parts of 5% iodine and glycerol) as used by Waerhaug and Steen.[2] In this latter group samples were also taken from gingival and tooth surfaces adjacent to the crevices investigated.

RESULTS

The results of the investigations of both the first and second groups are summarised respectively in Tables I and II.

Of the 63 crevices investigated by

TABLE I
Bacteriological Examination of Clinically Healthy Human Gingival Crevices

Site	*Type of Culture*	*Number Negative*	*Number Positive*	*Total Number of Crevices*
From crevice	Aerobic	5	58	63
	Anaerobic	3	43	46
Controls (from tube alone)	Aerobic	33	3	36

aerobic culture 58 gave a growth of organisms. Similarly 43 out of the 46 crevices cultured anaerobically gave a growth. However, in the 33 control cultures only three gave a positive culture.

The difference between the numbers involved in each category of each group is highly significant ($p < 0.001$).

In the second group of crevices, studied following the application of iodine glycerol mixture, only two out of twenty-one crevices gave a positive culture. The samples from adjacent tooth and gingival surfaces were all negative, giving an indication of the effectiveness of this antiseptic. The results in this group closely parallel those of Waerhaug and Steen.[2]

DISCUSSION

The principal difficulty in a study of this nature is, of course, to obtain a bacteriological sample from the gingival crevice without contamination from adjacent tooth or gingival surfaces. Waerhaug and Steen[2] in their studies sought to overcome this difficulty by using an iodine glycerol mixture to sterilise these surfaces before entering the crevice with a steel blade to collect material. The reason given for supposing that this antiseptic does not penetrate the crevice is that, following the introduction, presumably to the deepest point of the "epithelial attachment,"[2,3] of concentrated masses of bacteria (soft deposits or concentrated bacterial cultures) this method gave positive cultures. It is our contention that in the shallower crevices this extremely effective antiseptic[10] had penetrated by capillary action and rendered the crevice sterile.

Krasse and Brill, (1960)[11] in their study of the effects of diet on the bacteria in the gingival crevice suggest that using this antiseptic it is easier to obtain negative rather than positive results, and also that repeated applications of antiseptic may render previously positive crevices negative.

Boyd and Rosenthal, (1958)[9] also found that the application of Iodine-glycerol mixture rendered the gingival crevice sterile and sought to overcome this difficulty by avoiding the use of an antiseptic in their study. However, these workers were aware

TABLE II
Bacteriological Examination of Clinically Healthy Human Gingival Crevices following the Application of Iodine-Glycerol Mixture

Site	*Type of Culture*	*Number Negative*	*Number Positive*	*Total Number of Crevices*
From crevice	Aerobic	19	2	21
From adjacent surfaces	Aerobic	7	—	7

of some of the difficulties of their method. Firstly, the insertion of the explorer into the crevice for the retraction of the free gingivae involves a risk of contamination from the tooth and gingival surfaces it contacts. Secondly, their control showed that in each case the explorer used to collect the sample was sterile, but it did not control contamination from adjacent tooth or gingival surfaces. Thirdly, in both methods a steel blade or explorer was used to collect the bacteriological sample.

The method devised for this study seeks to overcome these difficulties. The use of an antiseptic is avoided, contamination being prevented mechanically by the sterile metal tube. The control used shows that the internal surfaces of the tube which contacts the paper point were not responsible for contaminating the tube and thus material collected must be from the depths of the crevice. And finally, an absorbent paper point was used to collect a larger and therefore more representative sample than is possible with metal instruments.

Although the control used did show that in a significant number of cases material was collected from the crevice and was not contaminated by the tube, and at all times care was taken to avoid the introduction of contaminants to the crevice, there is a possibility that the placing of the sterile tube on the carefully dried gingival margin could have contaminated the crevice. Against this rather remote possibility we have not been able to devise an entirely effective control suitable for use on human subjects.

The non-inflammatory defence mechanisms of the gingival crevice have been discussed elsewhere[5, 12] and these factors are no doubt responsible for the removal of bacteria from the crevice. However, the entry of organisms into the crevice is favoured by the capillary action of the crevice, by the trauma and movement of mastication, and by the constant bathing of the crevice by saliva with its extremely numerous organisms.

Although the possibility of a resident crevicular flora cannot be discounted, it would appear probable that a state of equilibrium exists between the entry of salivary organisms and the defence mechanisms tending to eliminate them from the gingival crevice. This view is in accordance with the present findings that viable organisms exist within the majority of clinically healthy gingival crevices. Indeed, due to the presence of bacteria on most other comparable body surfaces it would seem unlikely, especially in the oral environment, to find such a situation sterile.

It seems likely that organisms penetrate as far apically as the coronal end of the "epithelial attachment" whereas in the more apical region bounded by the "epithelial attachment," which is considered by some to be a part of the gingival crevice or pocket,[2, 3] the entry of bacteria and antiseptics may be prevented by the adhesion[3] or attachment[6] of the "epithelial attachment" to the tooth surface.

SUMMARY

A method of bacteriological investigation of the gingival crevice is described.

Doubt is cast on the belief that the gingival crevice, free from calculus or deposits, is, as a rule, sterile.

By the described method the clinically healthy gingival crevice appears to contain micro-organisms in the majority of cases.

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Healing of Naturally Occurring Periodontal Pockets: Preliminary Report of the Effects of External Pressure

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FOLLOWING temporary surgical detachment of periodontal tissues from human vital teeth an epithelial lining grows down to a point where healing root and gingivae had been in close contact.[4] It grows past root areas previously gouged with a bur and stops where the gouges become shallow. This holds true whether these shallow surfaces are composed of either cementum or dentin.

The connective tissue union appearing at these shallow points prevents further epithelial invasion. More apical areas, regardless of the presence or size of root gouges or nicks, may thus leisurely complete the healing process with formation of new cementum and periodontal fibers.

These observations were explained by reasoning that there is a possible relationship between clot size and the time required for formation of connective tissue cells adjacent to and capable of union with the root. Since the clot is the size of the gouge it fills, the process probably is slower in the deeper gouges. If it is slow enough then epithelium grows down by default.

This interpretation suggested, in turn, that failure to obtain reattachment in naturally occurring pockets might, in part, be due to too large a blood filled space between root and flaccid gingival pocket wall. With this in mind, a previously unsuccessful attempt to secure healing of naturally occurring pockets[3] was repeated, but this time minimizing the blood clot thickness by pressing soft tissue against tooth. This paper is a preliminary report of the results.

PROCEDURE

The material consists of three maxillary anterior teeth taken from apparently healthy adults for prosthetic reasons. Pockets were present on the labial surfaces.

Tooth nicks were made, one at the bottom of the pocket (N_2) and the other on the crown as a fixed point for measurements. The nicks were started with burs

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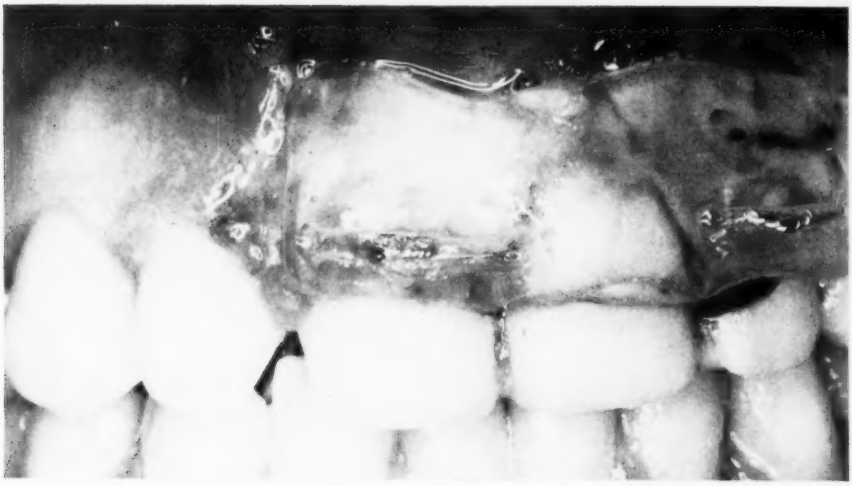

Fig. 1. Plastic stent relined with self curing acrylic, held firmly to teeth with wire ligatures in order to secure firm apposition of healing pocket wall to root.

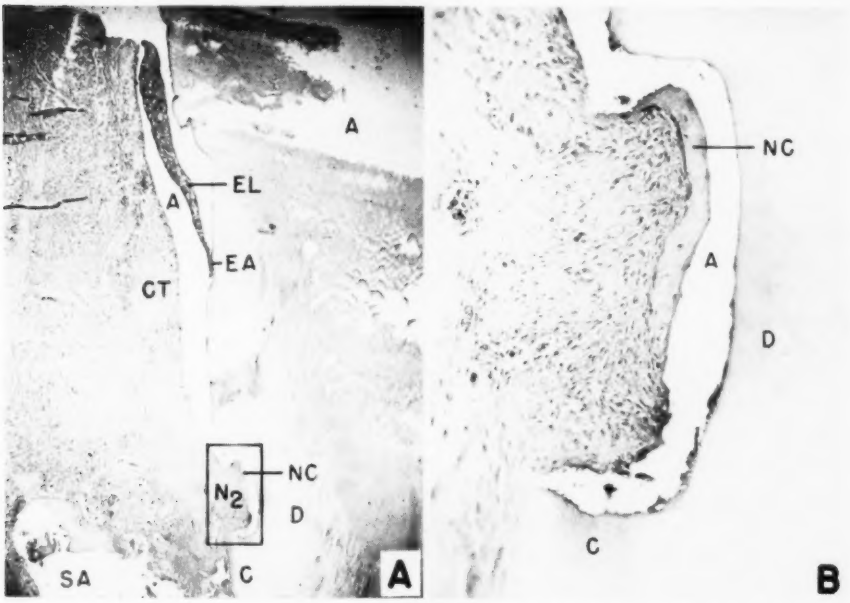

Fig. 2A. (Case #1—28 days postoperatively.) The epithelial lining (EL) terminates at the epithelial attachment (EA) about halfway down the pocket wall. A connective tissue surface (CT) remains apical to the epithelium, separated from the root by processing artefact (A). A nick (N_2) made close to the bottom of the pocket extends into the dentin (D). New cementum (NC) has formed in N_2. SA is a surgical artefact produced in the biopsy procedure.

Fig. 2B. This is a higher magnification of the inset in Fig. 2A. New cellular cementum (NC) can be plainly seen, separated by artefact (A) from the dentin (D) on which it has been deposited.

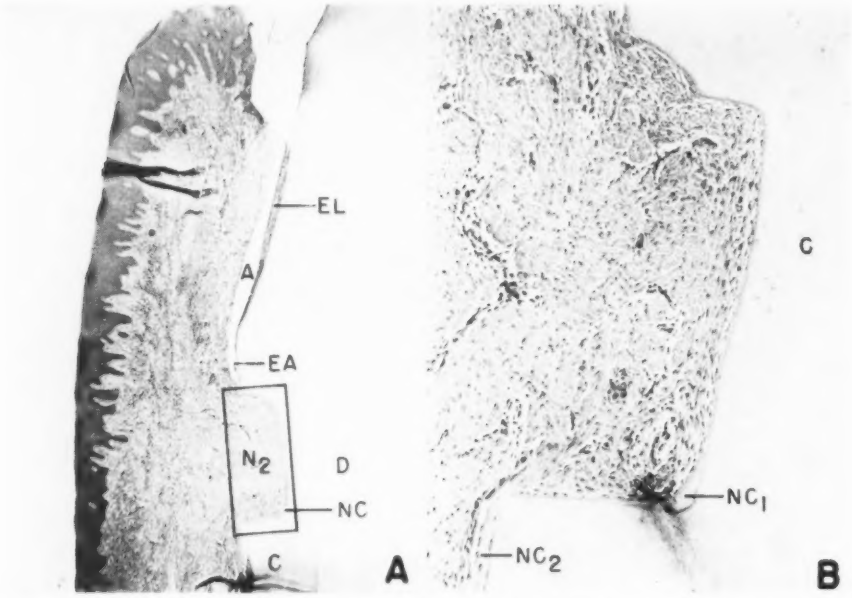

Fig. 3A. (Case #2—70 days postoperatively.) The epithelial lining (EL) halts at the epithelial attachment (EA) where the root deformity becomes shallow. The nick (N_2), near the bottom of the original pocket, is entirely within the original cementum (C). A pink amorphous deposit (NC) lines the exposed cemental surface.

Fig. 3B. This is a higher magnification of the inset in Fig. 3A. Relatively acellular new cementum may be seen in the nick (NC_1) and on the outer surface of the pre-existing cementum (NC_2).

and completed with small operative hatchets.

Attachment and lining epithelium were removed by curettage followed by excision of about 1.0 millimeters of the gingival rim and concluded with warm water lavage.[2]

The root was then curetted and cementum removed from the coronal half of the pocket with a bur. In some instances another groove or nick was made halfway up the pocket wall.

A previously prepared plastic labial stent (Fig. 1) was then relined with self-curing acrylic to secure very close adaptation of soft tissue to root. The stent was held firmly in position by .010 stainless steel orthodontic wire which encircled the teeth and passed through holes in the stent to be tightened on its outside surface. This arrangement was removed after one week.

Teeth and labial periodontal tissues were removed en bloc, as previously described,[3] in from four to ten weeks postoperatively.

The extracted masses were fixed in 10% formalin, decalcified and embedded in paraffin. Serial sections were then stained with hematoxylin and eosin.

RESULTS

All three cases (Figs. 2A, 2B, 3A, 4) showed epithelial linings invading part of the pocket length and stopping at shallow points in the root deformities. They also showed a connective tissue union apical to those points, and deposition of new cementum in the terminal nicks on both dentinal and cemental surfaces.

DISCUSSION AND SUMMARY

The terminal nicks were within the original pockets since they were made before the removal of epithelial attachment and

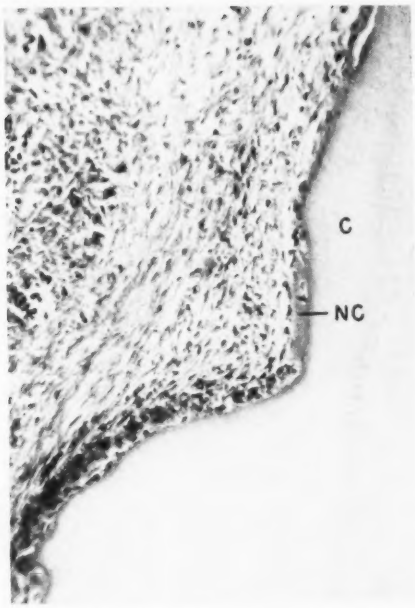

Fig. 4. (Case #3—35 days postoperatively.) This is another example of new cementum (NC) deposited on the surface of cementum (C) in the terminal nick in a naturally occurring periodontal pocket.

lining. The significance of this preliminary report rests on the demonstration that it is possible, in a naturally occurring periodontal pocket, to affect a connective tissue union between root and gingivae and to deposit new cementum on either dentin or cementum.

This evidence, presently confined to the more apical portions of pockets, is identical with the results in surgically created pockets.[4] It reinforces the concept that close contact is an important feature in this healing process. It also suggests that a smooth unmarred root surface and external pressure both may help to achieve this contact.

The use of immobilization and pressure as an aid to healing is as old as man himself. It has received particular emphasis in skin grafting where blood clots can be responsible for failure. Kazanjian and Converse[1] state that "without adequate pressure a collection of blood and serum forms between graft and bed." To obviate this, various kinds of pressure bandages and stents have been devised.

It is interesting to note that Rockoff, Rockoff and Sackler[5] presented a case of apparent reattachment in which an acrylic stent was worn for four weeks "to stabilize the tooth through support by the adjacent teeth." Although intended for stabilization this splint probably also held the healing tissues close together.

A more extensive report will be made upon completion of cases now being treated with improved pressure technics.

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BOSTON UNIVERSITY DEPARTMENT OF STOMATOLOGY

The following courses are offered by the Department of Stomatology Division of Graduate Studies, School of Medicine, Boston University and the Massachusetts Memorial Hospitals: October 30-November 3, 1961—Occlusal Adjustment, November 6-November 10, 1961—Periodontal Therapy.

A Histochemical Study of Periodontal Disease in the Maxillary Alveolar Processes of 135 Autopsies

by HAROLD M. FULLMER,* *Washington, D. C.*

PERIODONTAL disease is characterized by a loss of alveolar bone and fibrous connective tissues of the periodontium resulting in the loss of teeth.[1,2,3,4,5,6] Previous histological studies have usually concentrated their attention on the loss of connective tissues incurred by the inflammatory process. In this study, sections stained with the peracetic acid-aldehyde fuchsin-Halmi method frequently revealed marked degradative changes in the fibrous connective tissues at a distance from the inflammatory focus, and that the major repair of periodontal lesions occurred inferior to this location. New data is presented on the distribution of oxytalan fibers in the human periodontium, and on the nature of their participation in periodontal disease. Also included is a review of some previous work on histochemical analyses of oxytalan fibers, and a discussion of collagen metabolism as related to periodontal disease.

Oxytalan fibers have been shown to be a distinct type of connective tissue fiber, and a normal constituent of periodontal membranes of man.[7,8,9,10,11,12] Present evidence indicates that they may be related to elastic fibers.[7,8,13] They develop in new reparative tissue of the periodontium as demonstrated by their genesis in dental granulomas and radicular cysts,[10] and in the supportive connective tissues of ameloblastomas,[14] but their possible role in periodontal disease has not been previously investigated.

MATERIALS AND METHODS

The material examined consisted of 135 maxillary alveolar processes with teeth in situ taken at autopsy from individuals who died of various causes. Their ages ranged from 5 to 72 years. The specimens usually contained only molar teeth, but occasionally the second bicuspids were taken. The specimens were usually fixed in Lillie's aqueous calcium acetate 10% neutral formalin,[15] but some were fixed in Carnoy's solution,[15] 95% ethanol and 70% ethanol. The specimens were then decalcified in 5% formic acid, divided into halves by a mesiodistal cut, dehydrated, blocked into paraffin, and sectioned at 6μ. A few were cut so that buccolingual sections were obtained. Also, some fresh frozen sections of gum stained with various methods were examined.

Staining Methods: Control sections were stained with hematoxylin and eosin. Elastic fibers were demonstrated by either the Taenzer-Unna orcein stain,[15] or the orcinol-new fuchsin method.[16] Oxytalan and collagen fibers were demonstrated by either the peracetic acid-orcein-Halmi,[8] or the peracetic acid-aldehyde fuchsin-Halmi method.[7] The peracetic acid was prepared according to Greenspan,[15,17] and was kept in the refrigerator when not in use. The aldehyde fuchsin was prepared according to Gomori;[15] aged 72 hours at room temperature before use; kept in a closed container when not in use; and discarded one week after preparation. Mucopolysaccharides were demonstrated by azure A (0.5% concentration in aqueous 0.2M phosphate buffer pH 4 for 10 minutes at room temperature), by the peracetic acid-aldehyde fuchsin-Halmi stain, and by Rinehart's dialyzed iron-picrofuchsin stain[15] modified as follows in order to effect greater specificity of the reaction: the dialyzed iron was diluted 1:4 with distilled water; the sections were limited to 5 minutes in the ferrocyanide mixture; and the cocchineal staining step was omitted.

Carbohydrates were identified by the periodic acid-Schiff hematoxylin stain.[15] Lillie's allochrome stain for connective tissues was also used.[15] The alloxan-Schiff

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stain was used for the identification of protein.[18]

Enzymatic Digestions: Enzymatic digestions were carried out as follows: testicular hyaluronidase* and hyaluronidase inhibitor (heparin) was used mg:mg hyaluronidase. β-Glucuronidase** and β-glucuronidase inhibitor (saccharic acid) was used at 0.01 to 01. M concentration. A few milligrams of penicillin and streptomycin were added to the incubation mixtures to prevent the growth of microorganisms.

RESULTS

Normal Periodontal Structures: To apprehend the structural alterations associated with periodontal disease and demonstrated by some of the stains used in this study, it is appropriate at this point to first visualize the normal. The application of the peracetic acid-aldehyde fuchsin-Halmi stain to these tissues allows a perception of periodontal tissues not achievable with other methods. Figure 1 is a photomicrograph of a normal periodontal membrane in the transeptal region stained with this method. Oxytalan fibers stain purple, and collagen fibers stain green to greenish-yellow. In this area oxytalan fibers insert into the cementum and extend outward with the collagen fibers while they gradualy decrease in size and terminate varying distances from the tooth. Sometimes they arborize before termination. They extend neither to adjacent teeth nor to adjacent bone. Others in this region remain unattached to the tooth. An interproximal zone of varying size thereby usually remains devoid of oxytalan fibers and composed of bundles of collagen surrounded by a mass of mucopolysaccharide(s).[19] In rare instances oxytalan fibers are seen in this area where they appear to evolve from the mass of mucopolysaccharide(s) as they do during embryogenesis.[11]

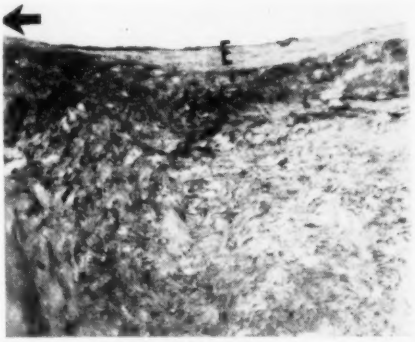

Fig. 4. Section of periodontal membrane distal to the upper third molar from a white male aged 20 who died subsequent to surgery for a malformation of the heart. The cemento-enamel junction is immediately to the left of the arrow. Note the concentration of oxytalan (dark staining) fibers immediately inferior to the cells of the epithelial attachment. X435.

In the gingiva, some oxytalan fibers insert in the cementum at the neck of the tooth and proceed with the free gingival collagen fiber groups while others course in various directions including circumscribing the tooth. The circular oxytalan fibers may extend into the transeptal regions (Figs. 2 and 3). Some oxytalan fibers insert into the basement membrane of the epithelial attachment and progress into the sub-epithelial connective tissues where they may or may not arborize before termination.[19] A concentration of oxytalan fibers is usually observed immediately inferior to the cells of the epithelial attachment irrespective of its location on the tooth (Fig. 4).

Oxytalan fibers residing in the regions of the alveolar crest, horizontal or oblique groups of collagen fibers may insert into either cementum or bone, and usually course varying distances with the collagen fibers of their respective groups (Fig. 5). None were observed to span the entire width of the periodontal membrane. Usually, more and larger oxytalan fibers are found on the tooth than on the bone side of the periodontal membrane. Other oxytalan fibers course in many directions, and the periodontal membranes of the de-

*(Nutritional Biochemicals Corp.) 1:1000 in 0.1 M phosphate buffer pH 6 for 24-48 hours at 37° C.[15]

**(Nutritional Biochemicals Corp.) was used 15 mg:50 ml 0.1 M acetate buffer pH 4.5 for 24-48 hours at 37° C.

ciduous dentition contain many large oxytalan fibers aligned in an apico-occlusal direction.[11, 19] In addition, some that appear continuous with the interior limiting membrane of lymphatics extend occlusally to anchor into the cementum of the tooth.[11] In the apical region, oxytalan fibers course in many directions and frequently form a network.[19]

In general, oxytalan fibers tend to be larger and more numerous in functional than in non-functional periodontal membranes; and portions of periodontal membranes subjected to greater stress contain larger and more oxytalan fibers than portions which are less functional. The degree of organization and orientation of the various components of the periodontal membrane indicate that it has progressed to a high state of differentiation.

The diffuse, amorphous substance throughout the periodontal connective tissues that stains purple with the peracetic acid-aldehyde fuchsin-Halmi method is presumed to be mucopolysaccharide since it is digested by β-glucuronidase; and the digestion is prevented by saccharic acid which is a known inhibitor of β-glucuronidase.[9] The mucopolysaccharide(s) is usually, but not always demonstrable with the periodic acid-Schiff method, and only occasionally with the Rinehart (Hale) method. Mucopolysaccharides reactive metachromatically with azure A are sometimes demonstrated in the periodontal membranes of the adult, but they are routinely demonstrated in these tissues during the developmental state.[11] The stainable component of oxytalan fibers is believed to be mucopolysaccharide(s) because it is removed by either testicular hyaluronidase, β-glucuronidase or by lysozyme if the fibers are previously oxidized by peracetic acid.

Alterations with Periodontal Disease: A critical analysis of numerous sections from each of the 135 specimens manifesting varying degrees of periodontal disease indicates that the decrement of connective tissues occurs via at least two mechanisms. Connective tissues were destroyed (a) in

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All photomicrographs are of sections stained with the peracetic acid-aldehyde fuchsin-Halmi method. Collagen, bone and dentin stain green to greenish-yellow; oxytalan fibers stain purple. Mucopolysaccharides also stain purple and appear as granular and amorphous masses. A,B,C, and D are zones of the periodontium which depict distinct types of connective tissue physiopathology; namely, inflammatory zone, normal zone, degradative zone, and repair zone, respectively. 1,2, and 3 refer to the first, second and third molars, respectively. AB, alveolar bone; CM, cementum; DG, degradative; E, epithelial attachment; N, normal; and R, repair.

Fig. 1. Section of periodontal membrane in the transeptal region distal to the upper left first molar from a white male who died of a carcinoma of the bronchus. Oxytalan as well as collagen fibers insert into the cementum and extend outward. X415.

Fig. 2. Section of periodontal membrane lingual to the upper left first molar at the clinical neck from a 36-year-old, white female who died of a choriocarcinoma. The teeth were in good occlusion, and the oral hygiene appeared good. Oxytalan fibers are concentrated near the tooth. The X's in Figures 2 and 3 are in identical locations and indicate the area of magnification of Figure 3. X275.

Fig. 3. A magnification of Figure 2 in the region of the X. Note that oxytalan fibers not only follow the main course of transeptal collagen fibers, but are seen cut on cross section which indicates a circumferential alignment. X355.

Fig. 5. Section of normal periodontal membrane from an upper left first molar in the region of the oblique group of periodontal fibers. Oxytalan fibers insert into either cementum or bone and thereafter frequently follow the course of collagen fibers. X430.

Fig. 6. Interdental region between the upper left first and second molars of a white male aged 19 who died postoperatively from surgery for a pulmonic stenosis. He had fair oral hygiene; his teeth were in normal occlusion; and a mild gingivitis was evident clinically. A, zone of inflammation; B, zone of normal connective tissues; C, zone of connective tissue degradation (see text and Fig. 7); and D, zone of repair (not evident at this magnification). X14.

Fig. 7. Zone C in another section taken from the same individual as Figure 6. Normal connective tissues persist in the upper part of the photomicrograph; however, varying degrees of connective tissue degradation (DG) in the absence of inflammation is noted. The degradation is identified by the loss of density, the acquisition of a purplish-gray stain, etc. (see text). X105.

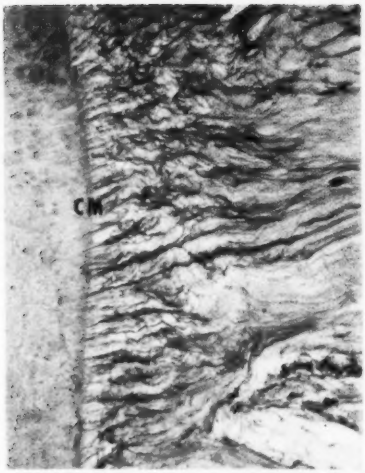


Fig. 1

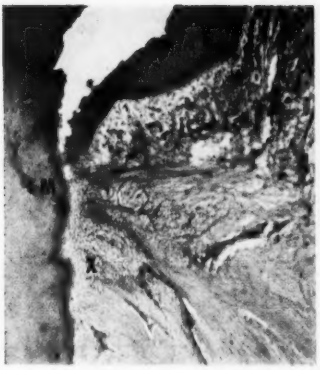


Fig. 2

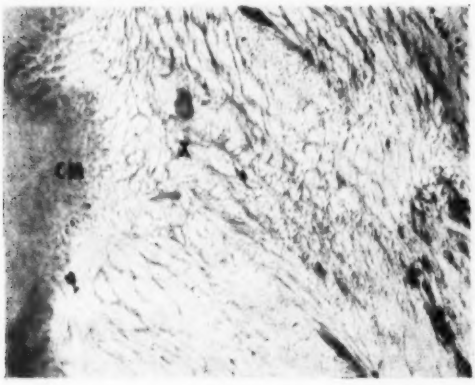
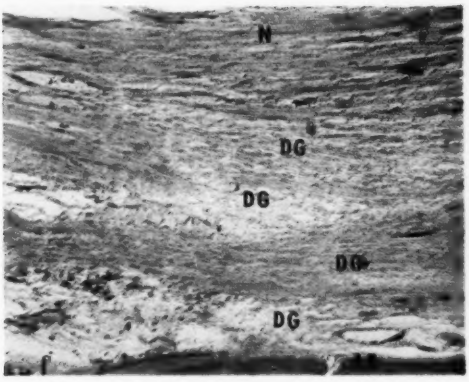


Fig. 3

Fig. 5

Fig. 6

Fig. 7



regions of inflammation, and (b) in regions apart from inflammation. An explanation and comprehension of the suggested sequence of events in this disease may best be achieved by a division of the periodontium into zones A,B,C, and D on the basis of the type of pathosis present. Although a wide variability in the magnitude of the zones in various cases was observed, the various zones of a typical early interproximal lesion of periodontitis are indicated in Figure 6 which was taken from between the first and second molars of a white male aged 19 who died following surgery for a pulmonic stenosis. A description of the zones in this and other lesions follows.

Zone A is characterized by inflammation. The extent of zone A varied widely depending upon the intensity of the inflammation; in severe cases, it included the entire periodontium. Inflammation was always situated in approximation to microorganisms and other debris in the gingival crevice. On the basis of the methods used in this study, it was impossible to adjudge whether the loss of tissue accrued (a) as a direct effect from the degradative enzymes and other noxious substances liberated from microorganisms, (b) secondarily from the inflammatory process, or (c) both. The region contained varying quantities and types of leukocytes depending upon the nature of the inflammatory process, and connective tissues were absent in areas wherein they accumulated. Many authors have described the loss of periodontal tissues associated with inflammation, and whereas the methods employed in this study did not disclose additional knowledge of this subject, this account will not be recapitulated here. Significant repair was observed in this region only if the inflammation was slight. Sections stained with the peracetic acid-aldehyde fuchsin-Halmi method indicated abundant mucopolysaccharide(s) in this area.

Zone B is a region of normal connective tissues, and is appropriately depicted in Figures 1 and 3. The presence of normal tissues at this site has been previously noted.[4,20] Figure 6 portrays the usual magnitude of zone B (between the bars) in cases manifesting a mild gingivitis. In a few cases exhibiting a very slight gingivitis (in zone A), normal connective tissues were observed throughout the rest of the periodontium. However, these were rare exceptions. Figure 8 is an interproximal lesion between the upper left first and second molars from a white female aged 33 who died of chronic glomerulonephritis, hypertension and cardiac failure. She had good oral hygiene and her teeth were in normal occlusion. Zone B in this case is represented by only a few remnants of normal connective tissue fibers indicated by arrows in Figure 8 and enlarged in Figure 9. In Figure 12, zone B is extinct. The latter is an interproximal lesion between the upper left first and second molars from a white female aged 28 who died of a malignant melanoma.

Reference to Figure 6 signifies that the periodontal connective tissues in zone C are situated apart from the inflammatory focus; however, they appeared in various degrees of degradation. The connective tissues are destroyed in this region in a manner different from that in region A. Microscopically, this type of loss is characterized by (a) the absence or extreme paucity of inflammatory cells, (b) a decreased density of the periodontal membrane which may even display spaces or vacuoles, (c) the acquisition of a purplish-gray stain by affected connective tissues with the peracetic acid-aldehyde fuchsin-Halmi stain, and (d) alterations in vascularity may or may not be evident; i.e. increased vascularity, dilated blood vessels or congestion. This peculiar type of pathosis was not demonstrated with other staining methods used. The extent of zone C also varies with individual cases and is dependent upon factors undetermined at this time. The observations that zone C may extend from the transeptal area to the apices of the teeth in cases manifesting only slight inflammation in zone A, and the converse, indicate that the magnitude of zone C is not always

proportional to the severity of the inflammation in zone A.

The initial degradative connective tissue lesions of the type seen in zone C appeared to consist of collagen and/or oxytalan fibers in varying degrees of dissolution with generally a concomitant influx of mucopolysaccharide(s) which stained purple to purplish-gray with the peracetic acid-aldehyde fuchsin-Halmi stain. Figure 13 is the transeptal interdental area between the upper left second and third molars from a white male aged 20 who died following surgery for a heart malformation. The arrows point to sites manifesting this very early stage of retrogressive connective tissue change. Figures 6 and 7 depict zone C in a typical case with a mild gingivitis. Note the various degrees of degradation (DG). Figures 8, 9 and 10 further characterize zone C in a case with a more intense inflammatory process in zone A; Figure 12 illustrates the severe zone C type degradation of connective tissues throughout the periodontium in a case exhibiting only slight gingivitis in zone A. In some instances, such as illustrated in Figures 8, 10 and 13, bone resorption in zone C appears to accompany the retrogressive connective tissue change. In other situations, such as exemplified by Figure 12, bone resorption succeeds the destruction of the fibrous connective tissues. Repair did not coexist in locations manifesting the zone C type of retrogressive change; the two phenomena appear to be mutually exclusive.

Zone D is characterized by repair. It was particularly noted above that a little repair was sometimes observed in zone A and none in zone C. Zone D may persevere in the transeptal regions, but it only rarely maintained this height. Its occlusal extent was usualy observed to be one to two millimeters inferior to the crest of the alveolus. Characteristically, the major part of the repair tissue that evolved consequent to periodontal disease proliferated from perivascular locations within the bone marrow (Figs. 8, 10, and 11). More perivascularly situated repair tissue was observed at the occlusal than at the apical extent of zone D. The repair tissue contained oxytalan as well as collagen fibers (Fig. 11). Coincident with the development of repair tissue near the occlusal extent of zone D was the degradation of connective tissues and resorption of bone at the apical extent of zone C. Figures 8, 9, 10 and 11 illustrate particularly well the concept that progressive resorption of the crest of the alveolar bone and degradation of the occlusally adjacent connective tissues concomitant with the expansion of repair tissue proliferating from perivascular sites within the bone marrow results in the gradual development of new periodontal membrane inferior to its former position. Likewise, in areas further apically situated, repair appeared to emanate from perivascular locations within the bone marrow, and to proceed in the above stated course. In a few cases the zone C type of lesion occupied the entire periodontal membrane and no repair was found (Fig. 12). Repaired periodontal membranes in new transeptal areas always appeared less functional than the original; i.e., collagen and oxytalan fibers were not developed to the normal degree, and collagen bundles were inadequately oriented.

The effect of the drugs which these individuals received, or the disease itself on the connective tissues of the periodontium is unknown.

DISCUSSION

Examination of the constitution of the periodontal membrane particularly as visualized by sections stained with the peracetic acid-aldehyde fuchsin-Halmi method reveals a distinctive connective tissue structure. It differs from skin and connective tissues generally by the absence of elastic and the presence of oxytalan fibers, by a large mucopolysaccharide content, and by a high degree of organization and orientation of the fibrous components. These qualities relate periodontal membranes closely to tendons and ligaments. It follows that true regeneration of periodontal membrane, like tendon, entails the pro-

duction of a highly evolved tissue. It is perhaps significant that Buck has found that the repaired portion of a tendon can always be discerned from the normal,[21] just as this investigation has revealed that new portions of periodontal membranes appear less developed and less organized than normal. Buck's work also suggested that mature tendon cells were incapable of division, and that new tendon collagen was produced by new connective tissue cells derived from tendon sheaths. Likewise, in this study it was apparent that the major repair tissue accrued from cells originating from perivascular locations within the bone marrow and not from cells of the periodontal membrane. The concept that the periodontal membrane is a highly differentiated organ cannot be overly stressed.

Some information about the metabolic behaviour of collagen acquired through diverse disciplines may assist us in an understanding of the behaviour of collagen in periodontal disease. For example, some data indicate the possibility of a retention of some collagen fibers throughout one's lifespan, and that subtle changes transpire with time rendering them less susceptible to deleterious influences. X-ray diffraction patterns of the human chordae tendineae of the heart disclosed an increasing degree of molecular orientation of collagen with increasing age of the individual.[22] The shrinkage temperature of collagen increases with increasing age of the individual,[23] and human collagen loses its osmotic swelling capacity and ability to swell at an elevated temperature with advancing age.[24, 25] Human skin collagen becomes less susceptible to collagenase digestion with advancing age.[26] Studies on the soluble collagens have revealed that (a) the type of reticulin fibers that develop in early granulation tissue and are characterized by selective silver impregnation stains are a form of soluble collagen,[27] (b) that newly formed collagen is easily extractible with weak salt solutions or even by cold water, but as the collagen ages, increasing salt concentrations are required for its solubilization;[28, 29, 30] and, (c) that mature collagen becomes insoluble.[28, 29, 30] A consideration of these qualities indicates that an increasingly organized, oriented and resistant collagen structure is achieved only after a period of years. These studies suggest that newly formed collagen may need protection from factors fostering its solution until substantial maturation has been accomplished.

Information about collagen maturation obtained from studies on the restoration of tensile strength of tendons after their scission also indicates that increasing maturation occurs with time. Mason and Allen found that dogs with severed Achilles tendons required 3 weeks' time for the development of sufficient tensile strength to permit the use of the extremity, and that the tensile strength gradually increased until the termination of the experiment at one year.[31]

The degradation of collagen and oxytalan fibers in zone C was regularly observed, and this thinning, which was frequently seen in the transeptal regions, is viewed ominously since this constitutes a weakened structure in advance of the inflammatory focus. Also, in some cases this type of lesion constituted the means whereby the major decrease of connective tissues occurred. A knowledge of the factors promoting the catabolic state of collagen and oxytalan fibers apart from an inflamed site are deemed essential for an understanding of the pathology of periodontal disease.

Theoretically, the application of appropriately labeled tracer techniques should divulge some information about situations fostering the anabolic or catabolic states of connective tissues. The use of C^{14} methylene labeled glycine revealed a relative metabolic inertia of tendon collagen in rats. Young rats rapidly incorporated the labeled glycine into collagen, but old rats incorporated only traces.[32] The turnover rate of collagen was concluded to be several hundred days. No experimental data on the turnover rate of collagen in man has been reported. The assumption that the

turnover rate in man is two to three years as in the rat may be hazardous, since this time interval represents the life span of the rat. A remarkably accelerated rate of production or loss of collagen may be superimposed on certain structures under physiological conditions. Harkness *et al* have demonstrated a six fold increase of collagen in the rat uterus within a 12 day interval during pregnancy, and its nearly total loss within *two* days after parturition.[33, 34, 35] They postulated that estrogen plus a "stretching effect" induced the uterine collagen formation. The operation of both factors was concluded from the observations that (a) uterine horns without a fetus contained only slightly increased collagen, (b) the quantity of collagen increased with each fetus, and (c) very little collagen was found in the placental stumps. They offered no mechanism to explain the precipitous loss of collagen consequent to parturition. Its loss by sloughing into the uterine and vaginal cavities was ruled out.

Another instance of an accelerated but less dramatic rate of collagen loss under physiological conditions is around erupting teeth.[36] Mature collagen of the oral mucosa was observed to be lost and the space usually simultaneously infiltrated with mucopolysaccharide(s) in advance of the erupting teeth. Interestingly enough, this pattern of connective tissue alteration was indistinguishable from that observed in some cases of periodontal disease in zone C. Pressure alone cannot account for the dissolution of collagen in advance of erupting teeth because the region immediately in advance of the erupting teeth always exhibited an anabolic state as indicated by new collagen and oxytalan fiber formation.[36] Engel has previously studied the alterations of ground substance associated with the eruption of the teeth, but he made no mention of changes in collagen.[37]

These data indicate that collagen is rapidly produced during the growth of the individual; that it is relatively (to other proteins) metabolically inactive once it is formed; that it may become increasingly resistant with time to forces facilitating its solution; and the mechanisms providing its maintenance are unknown. Its production may be stimulated by injury or by certain physiological situations cited above.

Fig. 8. Section of the interdental area between the upper left first and second molars from a white female aged 33 who died of chronic glomerulonephritis, hypertension and cardiac failure. She had good oral hygiene and her teeth were in normal occlusion. A moderate inflammation is present in zone A in apposition to the colonies of microorganisms. Zone B is represented by only a few shreds of normal transeptal fibers indicated by the arrows. Zone C is prominent. Regions 9 and 10 of zone C are magnified in Figures 9 and 10 respectively. Zone D includes the rest of the periodontium. Regions labeled R exhibit repair proliferating from perivascular locations. X14.

Fig. 9. A magnification of region 9 in Figure 8. Some normal periodontal membrane remains in the upper part of the photomicrograph, but the typical zone C type of retrogressive connective tissue changes (such as areas labeled DG) prevails inferiorly; namely, loss of density, vacuolization, absence of inflammation and the acquirement of a purplish-gray stain. X120.

Fig. 10. A magnification of region 10 in Figure 8. Connective tissues are undergoing degradation (DG) in zone C in the upper right portion of the photomicrograph. Some normal periodontal membrane persists in the upper left. Proliferation and expansion of repair tissue at R is observed concomitant with bone resorption indicated by arrows. The area in the region of R subsequently develops into transeptal periodontal membrane. X90.

Fig. 11. A magnification of region R in Figure 10. The new reparative tissue contains collagen and oxytalan fibers. One of the latter fibers is indicated by the arrow. X415.

Fig. 12. Section of periodontal membrane between the upper left first and second molars from a white female aged 28 who died of a malignant melanoma. Zone A manifested only a slight gingivitis microscopically. Zone B is missing. The zone C type of connective tissue degradation (DG) prevails throughout the periodontium. A slight indication of repair (zone D) was observed at the apex of one of the teeth. X14.

Fig. 13. Section of the transeptal area between the upper left second and third molars from a white male who died postoperatively from surgery for a heart malformation. There is no zone A (no inflammation). Nearly all the connective tissues are normal except those areas indicated by the arrows which depict a very early stage of connective tissue degradation characteristic of zone C. There is simultaneous resorption of alveolar crest bone.

Fig. 8

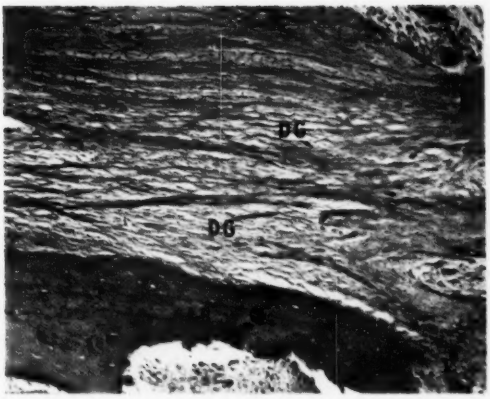


Fig. 9

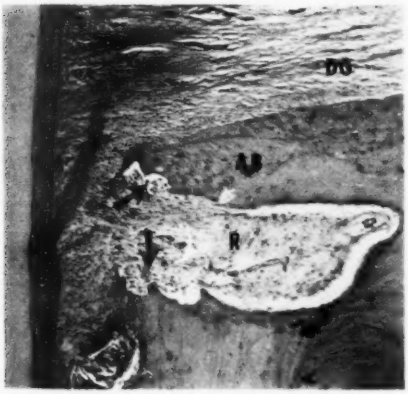


Fig. 10

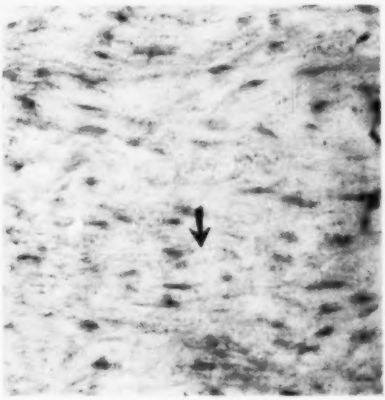


Fig. 11

Fig. 12

Fig. 13

SUMMARY

This report presents a histochemical analysis of periodontal disease in 135 maxillary alveolar processes with teeth in situ taken at autopsy. The periodontium was divided into zones according to the type of pathosis; namely, zone A, inflammation, zone B, normal connective tissues; zone C, degradative connective tissues; and zone D, repair.

The degradation of connective tissues and bone resorption in zone C which occurred in the absence of either inflammation or repair was extensively studied.

The major repair tissue consequent to periodontal disease occurred in zone D and evolved from perivascular sites in the bone marrow. New periodontal membrane results from the concurrent actions of connective tissue degradation and bone resorption in zone C, and the proliferation and expansion of repair tissue in zone D.

The peracetic acid-aldehyde fuchsin-Halmi stain is very useful for the study of the connective tissues of the periodontium for it not only demonstrates normal collagen, oxytalan fibers and mucopolysaccharides, but also a degradative phase of connective tissues observed in zone C.

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OFFICERS ELECTED AT THE ANNUAL MEETING OF THE AMERICAN ACADEMY OF ORAL PATHOLOGY

At the 15th annual meeting of the American Academy of Oral Pathology April 6-8 in Ann Arbor, Michigan, the following officers were elected for the year 1961-62: President, David F. Mitchell; President-elect, Norman H. Rickles; Vice-President, George Greene; Editor, Donald A. Kerr. Council members include Joseph L. Bernier, Barnet M. Levy, Hamilton B. G. Robinson, William G. Shafer, Harold R. Stanley and Richard W. Tiecke.

Robert J. Gorlin was elected to Secretary-Treasurer for a three-year period. George Greene and Richard Tiecke were elected as Examiners to the American Board of Oral Pathology.

The 1962 meeting will be held in Indianapolis, the 1963 in Miami and the 1964 at the National Institute of Health, Bethesda.

THE AMERICAN BOARD OF ORAL PATHOLOGY

Certification Examination

The next examination of the American Board of Oral Pathology will be given on December 9 and 10, 1961. Candidates will be notified of the place upon being accepted for the examination. All applications must be completed by August 1, 1961, for the December examination. Information and application blanks may be obtained from the secretary, Dr. William G. Shafer, Indiana University School of Dentistry, 1121 West Michigan Street, Indianapolis 2, Indiana.

The Influence of Psychiatric Disturbances on the Severity of Periodontal Disease

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THE relationship of psychic disturbances to the etiology of periodontal disease has been suggested repeatedly in the literature. Occlusal neuroses, neglect of oral hygiene, increased bacterial flora, lowered resistance to infection, qualitative and quantitative changes in the saliva, vascular changes, impairment of cellular nutrition, altered blood calcium levels, defective carbohydrate metabolism and endocrine disorders all are cited as possible etiologic factors in the emotionally disturbed individual which might produce periodontal disease.[1-13] Recently excessive periodontal disease was reported in a group of mentally subnormal children and in another group of mongoloid patients.[14, 15] It was the purpose of this study first, to confirm or deny on an epidemiologic basis the validity of the hypothesis that psychic influences predispose to the development of periodontal disease and second, to examine the nature of any associations found to exist.

SAMPLE

Two groups of male, veteran outpatients between the ages of 20 and 65 years with known medical histories were selected at random for a periodontal examination. Patients having less than six teeth were omitted from the study. The experimental group consisted of 104 patients who were receiving psychiatric treatment at the outpatient clinic of the Veterans Administration West Side Hospital, Chicago, Illinois. The control group consisted of 122 patients who were receiving treatment for conditions other than psychiatric disturbances at the same clinic (Table I). The diagnostic categories listed are those contained in the "International Classification of Diseases."[16]

Both groups of patients for the most part were employed and were living what otherwise might be classified as a "normal existence" except for the disabilities noted. It was felt that such a sample would yield more reliable data than one selected from a hospitalized population where many of the psychiatric patients are "out of contact" or the medical patients are seriously ill. No attempt was made to evaluate the possible effect of various systemic diseases on the periodontal disease experience of the control group, although this has been the subject of other published reports, which indicate that generalized diseases tend to increase the prevalence of periodontal disease.[17, 18, 19] For the purpose of this study the control group served simply as a convenient group, selected at random and presumed to have social and economic backgrounds similar to the psychiatric group with which the periodontal condition of the psychiatric group could be compared.

METHOD

The periodontal status of each patient was scored by the method of Russell,[20] by one examiner (O.P.G.), using a mouth mirror and periodontal probe under good lighting. No dental radiographs were taken. By the method of Russell, the periodontal status of each tooth is evaluated as follows: 0 = negative, 1 = papillary gingi-

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TABLE I

Primary Psychiatric and Medical Diagnoses by Age Group

Primary Diagnoses	Age Group						
	20-30	31-35	36-40	41-45	46-50	51-65	Total
Control Group							
Infective		2		4			6
Endocrine		3	2	1		1	7
Blood	1						1
Nervous System	4			2		1	7
Circulatory	2	4	2	3	1	5	17
Respiratory	2	2	2	1	4		11
Digestive	5	1	4	5	1	1	17
Genito-urinary			1	1	1		3
Dermatological	1	6	3	2			12
Bones & Joints	4	2		3		2	11
Ill Defined	5	5	4	3	5	1	23
Accidents	2	4		1			7
Total	26	29	18	26	12	11	122
Experimental Group							
Anxiety Reaction	7	6	14	14	8	3	52
Schizophrenic R. (chronic undiff.)	6	9	5	8	2	1	31
Schizophrenic R. (paranoid type)	2	3	2	1	1		9
Conversion R.			2	2			4
Depressive R.			2			1	3
Obsessive Comp. R.	1	1					2
Manic Depressive		1			1		2
Chronic Brain Syn.		1					1
Total	16	21	25	25	12	5	104

TABLE II

Relation of Psychiatric Disturbances to Severity of Periodontal Disease

AGE	GROUP	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
20-30	Control	26	21	1.04	3.5***
	N.P.	16	15	2.22	
31-35	Control	29	24	1.45	2.3*
	N.P.	21	20	2.44	
36-40	Control	18	14	1.23	1.9
	N.P.	25	24	2.15	
41-45	Control	26	21	1.04	5.6***
	N.P.	25	24	2.61	
46-50	Control	12	9	1.05	3.7***
	N.P.	12	13	3.47	
51-65	Control	11	9	2.59	0.9
	N.P.	5	5	3.42	
20-65	Control	122	100	1.30	6.9***
	N.P.	104	100	2.54	

*Exceeds 5% level of significance. **Exceeds 1% level of significance.

***Exceeds 0.1% level of significance.

vitis, 2 = marginal gingivitis, 4 = radiographic evidence of alveolar bone resorption (not used in this study), 6 = gingivitis with pocket formation, 8 = advanced alveolar destruction with mobility. The sum of the numerical values for all teeth in the mouth is divided by the number of teeth present to arrive at the Russell's score for each patient.

The vital statistics recorded included the psychiatric or medical diagnosis, frequency of brushing, degree of calculus, bruxism and clenching habits, income and educational levels and degree of anxiety.

FINDINGS

Experimental vs. Control Group. The experimental group of psychiatric patients showed higher periodontal scores than the control group in all age categories. (Table II). The differences were statistically significant up through 50 years of age. Over 50 years the difference was in the same direction but was not significant due presumably to the small number of patients in that category. In other words the findings confirm the hypothesis that psychiatric disturbances and periodontal disease are associated. The nature of that association was evaluated by analyzing the effect of several variable factors on the periodontal scores in the two groups.

Brushing Frequency. A clear-cut trend was apparent for the severity of periodontal disease to decrease as brushing frequency increased although the decrease was not statistically significant in either group. (Table III). When the severity of periodontal disease in both groups having the same daily brushing frequency was compared, the psychiatric group showed significantly higher scores than the control group. (Table IV). It also should be noted that the percentage of patients reporting the same brushing frequency was about the same in both groups, ruling out differences in brushing frequencies as a possible factor producing the difference in severity noted in the two groups. These findings clearly indicate that the higher periodontal scores in the psychiatric group were not associated with brushing frequency and that some factor other than brushing frequency was producing the significant differences noted.

TABLE III

Relation of Daily Brushing Frequency to Severity of Periodontal Disease

GROUP	BRUSHING FREQUENCY	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
CONTROL	0	17	16	1.75	1.9
	1	58	54	1.20	1.3
	2	31	29	1.07	1.4
	3	2	2	.96	
N.P.	0	17	17	3.47	1.0
	1	41	42	2.39	1.2
	2	29	29	2.13	1.1
	3	11	11	2.08	

TABLE IV

Relation of Same Daily Brushing Frequency to Severity of Periodontal Disease

BRUSHING FREQUENCY	GROUP	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
0	Control	17	16	1.75	4.7***
	N.P.	17	17	3.47	
1	Control	58	54	1.20	4.2***
	N.P.	41	42	2.39	
2	Control	31	29	1.07	3.3***
	N.P.	29	29	2.13	
3	Control	2	2	.96	1.6
	N.P.	11	11	2.08	

*Exceeds 5% level of significance.

**Exceeds 1% level of significance.

***Exceeds 0.1% level of significance.

Degree of Calculus. The severity of periodontal disease increased significantly in

TABLE V

Relation of Degree of Calculus to Severity of Periodontal Disease

GROUP	DEGREE OF CALCULUS	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
CONTROL	None	14	11	.60	1.2
	Mild	55	45	.94	5.3***
	Moderate	37	30	2.21	2.6**
	Heavy	16	13	3.45	
N.P.	None	10	9	.97	2.1*
	Mild	33	32	1.51	8.7***
	Moderate	43	42	2.76	4.0***
	Heavy	18	17	4.59	

*Exceeds 5% level of significance.

**Exceeds 1% level of significance.

***Exceeds 0.1% level of significance.

TABLE VI
Relation of Bruxism and Clenching to Severity of Periodontal Disease

GROUP	BRUXISM & CLENCHING	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
Control	Yes	42	34	1.27	.8
	No	80	66	1.46	
N.P.	Yes	53	51	2.57	.4
	No	51	49	2.44	

both groups of patients as the degree of calculus increased. (Table V). This direct correlation is in keeping with prevailing concepts concerning the role of calculus in the etiology of periodontal disease. It was surprising, however, to find more "moderate" and "heavy" calculus in the psychiatric group than in the control group —42 and 17 per cent as compared with 30 and 13 per cent. This latter finding suggested that differences in amount of calculus might be causing the difference in severity of periodontal disease in the two groups since the data clearly shows that calculus is a factor in producing higher periodontal scores. In order to evaluate this possibility, the average periodontal scores in both groups were compared while holding constant the degree of calculus and brushing frequency. When this was done the psychiatric group continued to show significantly higher scores than the control group, indicating that some factor in addition to calculus and brushing habits

DAILY BRUSHING FREQUENCY	None	Mild	Moderate	Heavy
3	C none NP .651	C .964 NP 1.980	C none NP 3.238	C none NP none
2	C .354 NP 1.414	C .813 NP 1.468	C 1.482 NP 2.658	C 3.084 NP 4.885
1	C .245 NP .676	C .969 NP 1.251	C 1.475 NP 2.670	C 3.716 NP 4.246
0	C .404 NP .750	C 1.087 NP none	C 1.492 NP 2.858	C 3.343 NP 4.858

DEGREE OF CALCULUS

Fig. 1. Average periodontal scores among 122 control and 104 psychiatric patients by degree of calculus and brushing frequency. The scores of the psychiatric patients are higher than the control in all twelve cells where comparisons are possible indicating a highly significant difference in the two groups of patients.

was producing the higher periodontal scores in the psychiatric group. (Figure 1).

Bruxism and Clenching. Bruxism and clenching were found to be more prevalent among the psychiatric than the control group of patients—51 per cent as compared to 34 per cent. (Table VI). This finding supports the concept that bruxism and clenching are manifestations of anxiety and emotional stress which are common symptoms of psychiatric disturbances. It was surprising, however, to find no significant difference in the severity of periodontal disease among those persons who bruxed and those who did not since brux-

FREQUENCY OF BRUSHING	None	Mild	Moderate	Heavy
3	Yes none No none	Yes .538 No 1.391	Yes none No none	Yes none No none
2	Yes .381 No .340	Yes 1.081 No .746	Yes .694 No 1.745	Yes none No 3.084
1	Yes .258 No .192	Yes 1.011 No .956	Yes 1.473 No 1.477	Yes 3.347 No 3.769
0	Yes 1.679 No none	Yes .896 No 1.229	Yes 1.190 No 1.694	Yes 7.333 No 1.348

DEGREE OF CALCULUS

Fig. 2. Average periodontal scores among 122 control patients who did or did not exhibit bruxism and clenching by degree of calculus and daily brushing frequency. Among those who did exhibit bruxism and clenching the scores are higher in five cells and lower in six cells indicating no significant difference.

ism and periodontal disease in the form of increased tooth mobility frequently are associated clinically, and since increased tooth mobility is one of the most heavily weighted criteria in Russell's scoring method. There continued to be no significant difference when the variable factors of calculus and brushing frequency were held constant. (Figures 2 and 3). These findings rule out bruxism and clenching as factors increasing the severity of periodontal disease in the two groups studied. The data also controvert the clinical impression that bruxism and periodontal disease are associated.

Educational Level. The severity of periodontal disease was found to decrease significantly in both groups of patients as

FREQUENCY OF BRUSHING	None	Mild	Moderate	Heavy
3	Yes .143 No 1.160	Yes 2.193 No 1.768	Yes 4.556 No 2.579	Yes none No none
2	Yes 1.037 No 1.791	Yes 1.458 No 1.478	Yes 2.624 No 2.715	Yes 4.696 No 5.454
1	Yes .480 No 1.069	Yes 1.445 No 1.019	Yes 2.796 No 2.465	Yes 4.215 No 4.276
0	Yes none No .750	Yes none No none	Yes 2.965 No 2.797	Yes 4.959 No 4.757

DEGREE OF CALCULUS

Fig. 3. Average periodontal scores among 104 psychiatric patients who did or did not exhibit bruxism and clenching by degree of calculus and daily brushing frequency. Among those who did exhibit bruxism and clenching the scores are higher in six cells and lower in seven cells indicating no significant difference.

educational level increased. (Table VII). This finding confirms a previous report relating social factors to the severity of periodontal disease.[21] The correlation between income level and severity of periodontal disease was not significant, however. In attempting to explain the correlation between educational level and the severity of periodontal disease, the average brushing frequencies at different educational levels were compared. When this was done a highly significant difference in brushing frequency was found between the "Grade 8" and "College" levels. (Table VIII). This finding suggests that increased brushing frequency played an important role in decreasing the severity of periodontal disease at the higher educational levels. Better brushing habits and more frequent visits to a dental office may contribute further to the decrease noted, although these factors were not measured.

Degree of Anxiety. A qualitative assessment of the degree of anxiety manifested by each psychiatric patient was made by the psychiatrist based upon an intimate

TABLE VII

Relation of Educational Level to Severity of Periodontal Disease

GROUP	EDUCATION	PATIENTS NO.	PATIENTS %	RUSSELL SCORE	CRITICAL RATIO
CONTROL	Grade 8	34	28	2.03	2.3*
	Grade 12	55	45	1.25	3.0***
	College	33	27	1.01	1.3
N. P.	Grade 8	22	21	3.14	1.4
	Grade 12	57	55	2.55	3.8***
	College	25	24	1.66	3.2***

*Exceeds 5% level of significance.

**Exceeds 1% level of significance.

***Exceeds 0.1% level of significance.

TABLE VIII

Relation of Educational Level to Daily Brushing Frequency

GROUP	EDUCATION	PATIENTS NO.	PATIENTS %	BRUSHING FREQUENCY	CRITICAL RATIO
CONTROL	Grade 8	34	28	.90	4.1***
	Grade 12	55	45	1.27	5.0***
	College	33	27	1.33	.5
N. P.	Grade 8	22	21	1.04	2.1*
	Grade 12	57	55	1.45	2.3*
	College	25	24	1.74	1.1

*Exceeds 5% level of significance.

**Exceeds 1% level of significance.

***Exceeds 0.1% level of significance.

FREQUENCY OF BRUSHING	None	Mild	Moderate	Heavy
3	1 - .143 2 - none 3 - 1.160	1 - 1.034 2 - 2.409 3 - none	1 - 2.579 2 - 4.556 3 - none	1 - none 2 - none 3 - none
2	1 - 1.115 2 - 1.037 3 - 2.467	1 - 1.451 2 - 1.460 3 - 1.607	1 - 2.770 2 - 3.122 3 - .823	1 - 3.704 2 - 5.191 3 - 5.454
1	1 - 1.069 2 - .480 3 - none	1 - .731 2 - 1.537 3 - 1.093	1 - 2.524 2 - 2.057 3 - 3.286	1 - 2.747 2 - 4.276 3 - 5.683
0	1 - none 2 - .750 3 - none	1 - none 2 - none 3 - none	1 - 3.175 2 - 3.140 3 - 2.338	1 - none 2 - 5.529 3 - 3.516

DEGREE OF CALCULUS

Fig. 4. Average periodontal scores among 104 psychiatric patients who exhibited 1) mild, 2) moderate or 3) severe degrees of anxiety by degree of calculus and daily brushing frequency. The scores for moderate or severe anxiety are higher than for mild anxiety in 10 out of 13 cells where comparisons are possible indicating a moderately significant relation between degree of anxiety and severity of periodontal disease.

knowledge of the case history of the patient. It was not practical to obtain this type of information for the control patients. When the variable factors of calculus and brushing frequency were held constant, the severity of periodontal disease increased significantly as the degree of anxiety increased. The periodontal scores for "moderate" or "severe" anxiety are higher than for "mild" anxiety in 10 out of 13 cells where comparisons are possible, indicating that degree of anxiety and severity of periodontal disease are related. (Figure 4).

DISCUSSION

The findings of this study clearly indicate that mental disturbances and periodontal disease are associated, and that association is due to some unknown intrinsic factor over and above the existence of possible poor oral hygiene habits, excessive calculus or occlusal neuroses. This unknown intrinsic factor apparently is closely associated with the degree of anxiety manifested by the patient and therefore probably is under the control of the autonomic nervous system. The close relationship between the central and autonomic nervous systems is well known. Vascular changes, tissue metabolism and endocrine function all are influenced by the autonomic system. It is reasonable to assume that the periodontal changes seen in the psychiatric patient are mediated through one or more of these processes. Recent experimental evidence has shown that marginal gingivitis and gastrointestinal lesions can be produced in dogs by stimulation of the hypothalamus which is the nerve center of the autonomic system.[22]

The greater amount of calculus found

among the psychiatric patients cannot be attributed to differences in brushing frequency since this factor was about the same in both groups. It is possible, however, that calculus formation may be related to a higher salivary pH in the psychiatric group. Salivary pH was not measured in this study although Starr[23] and Rich[24, 25] have demonstrated a relationship between emotional changes and pH of the saliva. In one series of tests the pH increased to an average of 7.20 during emotional excitement returning to an average of 6.73 after the subjects had become calm. One theory of calculus formation attributes the precipitation of calcium phosphate to the elevation of the pH of the saliva.[26, 27, 28]

The fact that no difference was found in the severity of periodontal disease among those persons who bruxed and those who did not suggests a possible need for revision of prevailing concepts concerning the role of bruxism in the etiology of periodontal disease. This finding is contrary to innumerable references in the literature that bruxism and periodontal disease have a cause and effect relationship. If this belief were true, one would have expected to find a difference in the periodontal scores among those who bruxed and those who did not.

SUMMARY

An experimental group of 104 psychiatric patients and a control group of 122 non-psychiatric patients, age 20-65 were examined to determine whether a difference in periodontal disease experience existed in the two groups and to examine the nature of any differences found to exist.

The findings are summarized as follows:

1. The severity of periodontal disease was significantly greater among the psychiatric than the control group of patients. Significant differences in severity persisted even when such variable factors as degree of calculus, brushing frequency and the habit of bruxism and clenching were held constant in the two groups.

2. Among the psychiatric patients, the severity of periodontal disease increased significantly as the degree of anxiety increased.

3. The severity of periodontal disease increased significantly in both groups of patients as the degree of calculus increased.

4. The severity of periodontal disease decreased in both groups of patients as brushing frequency increased although the decrease was not statistically significant.

5. The habit of bruxism and clenching was not related to the severity of periodontal disease in either group of patients although the habit was more common among the psychiatric group.

6. The severity of periodontal disease decreased significantly in both groups of patients as the educational level increased.

7. Brushing frequency increased significantly in both groups of patients as educational level increased.

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Robert L. Roseberry, D.D.S.

San Francisco, California

April 25, 1961

Members of the American Academy of Periodontology will be saddened to learn that Dr. Robert L. Roseberry, prominent periodontist in San Francisco, died April 25, 1961, at the age of 57. Dr. Roseberry was past-president of the California Academy of Periodontology and was also active in the American Academy of Periodontology. He was widely known for his excellent clinics and papers which he presented at dental meetings.

Electron Microscopy of Periodontal Structures

by NORMAN L. ESTERL, D.D.S., M.SC.,* *Columbus, Ohio*

THE electron microscope has been used, along with other methods, to advance our knowledge of the teeth and investing tissues. This study of the developing molar root of the rat confirmed in part, previous descriptions of osteoblasts and bone matrix. In addition the cytology of the cells from the developing periodontal ligament and an osteocyte is described.

MATERIALS AND METHODS

Mandibular first molars of Wistar and Sherman strain white rats were used in this experiment. Their ages varied from 18 to 22 days. The developing roots were amputated at the cervix. Care was taken to preserve the periodontal ligament and surrounding bone.

The roots were dissected under a microscope; physiological salt solution was used to help retard post-mortem degeneration.

The specimens were fixed in isotonic 1% osmium tetroxide, pH 7.25 – 7.3 for 1 to 8 hours and were dehydrated with 70%, 95% and 100% alcohols respectively.

All specimens were embedded in a mixture of butyl and methyl methacrylate (80:20) according to standard electron microscopy techniques.[1,2] At the time of embedding the specimens were oriented so that the ribbon of sections obtained on microtomy was cross-section of the developing root.

Ultrathin sections were obtained with a Porter-Blum microtome equipped with a diamond knife. The ribbons were allowed to spread by floating them on a warm solution of 10% acetone and 90% distilled water. The sections were then picked up on carbon-coated collodion 200-mesh copper grids and examined under the electron microscope, Type RCA EMU 2B.

This method did not permit the study of the developing periodontal ligament, cementum and bone in one thin section. Frajola, et al[3] and Gay and Anderson[4] described techniques which permitted a study of serial sections. This was modified by transferring sections immediately from the sectioning trough to a Petri dish containing warm acetone and water. After allowing fifteen minutes for the thin sections to spread, those with a bluish-gray cast were collected on wire loops. This technique made it possible to examine a thin section showing periodontal ligament and alveolar bone.

A circular revolving mechanical stage microscope was used to orient the thin sections collected on the wire loops. The developing bone and dentin was first identified under a 3.5-power objective lens, than a 10-power objective lens was used for more detailed orientation of the specimen and grid.

The Sjostrand-type Athene grid provides suitable slots to view a section either vertically or horizontally without obstruction from the grid bars.

OBSERVATIONS

Electron micrographs of developing alveolar bone show the osteoblast to be a flat, elongated cell. They appear to form a layer and overlie one another like shingles. The nucleus lies in the thicker end of the cell (Figure 1).[5]

The cytoplasm of the osteoblast also

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*Captain, USAF (DC). Assigned as graduate student in periodontology, Ohio State University. The contents of this article are the personal views of the author and are not to be construed as statement of official Air Force Policy nor as Air Force indorsement of any commercial product described. This paper is based on the thesis presented in partial fulfillment of the requirements for the degree of Master of Science, Ohio State University, Columbus, Ohio, awarded December 18, 1959.

The experiments were conducted according to the "Rules Regarding Animal Care," as established by the American Medical Association.

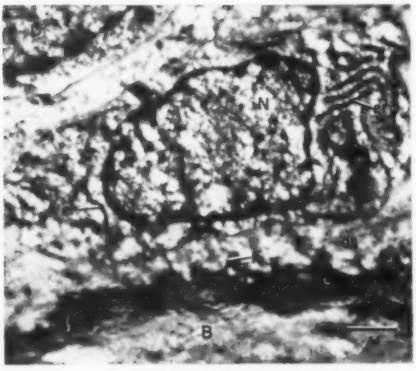

Fig. 1. Electron micrograph of an osteoblast (O) approximating bone. The nucleus (N) and endoplasmic reticulum (er) are seen in the cytoplasm. Between the bone surface (B) and the osteoblast is the osteoid (os). Fibers (F) project into the osteoid from the bone surface. A nucleus from a neighboring cell is visible in the upper left-hand corner of the micrograph (C). Mag. 12,500X.

tends to follow the contour of the developing bone. In Figure 2 the cell is seen to dip into a depression on the bone matrix surface.

Mitochondria are present in the cytoplasm and appear as short rod-like bodies (Figure 2). Numerous other mitochondria are present but sectioned at various angles. Fibers project from the developing bone into the bone matrix (Figure 3). Also the cut ends of fibers can be seen in the intercellular substance when they are cross-sectioned.

Endoplasmic Reticulum: Dispersed throughout the cytoplasm of osteoblasts are paired lamella whose membranes form a tubular-like system (Figures 1, 3, 4). These tubular structures interconnect with one another and form a part of the cytoplasm of most cell types.[6] The membrane walls appear parallel in cut sections, but when sectioned across their long axis, they appear tubular. The structure and amount of endoplasmic reticulum present in the cytoplasm of cells varies with different cell types.[7]

The inner surface of the endoplasmic reticulum is smooth but the outer surface is seen to have many small dense granules (Figure 3).[8] They are sometimes known as ergastoplasmic sacs, small particulate granules and Palade's granules. These granules appear to be "attached" to the outer surface of the endoplasmic reticulum. The endoplasmic reticulum may be continuous with the outer nuclear membrane.[9] In Figures 4 and 5 it is seen that the walls of the endoplasmic reticulum are connected with the nuclear membrane. Also dispersed throughout the cytoplasm are many dense granules of various sizes.

Osteocyte: The osteocyte shown in Figure 6 is probably an immature one. The nucleus is oval in shape in contrast to the flattened nuclei of osteocytes of mature lamellated bone.[10]

The osteocyte completely fills the lacuna. The cell membrane is evident and protoplasmic processes can be seen projecting from the cells as a dense fiber-like structure. Other canaliculi in cross-section show the protoplasmic process has a dense center.

The outer nuclear membrane is seen to project from the nucleus. The cytoplasm is finely granular and contains other unidentified components. Of particular interest is the decreased amount of endoplasmic reticulum.

Developing Periodontal Ligament: Numerous cellular elements are found in elec-

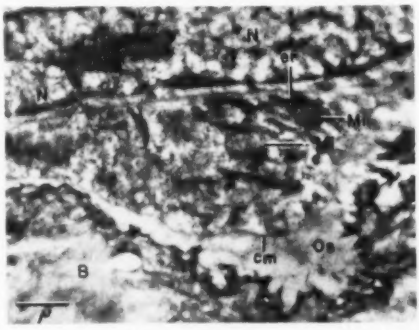

Fig. 2. Osteoblast (O) following the contour of the bone (B) surface. Os—osteoid; M—mitochondrion; er—endoplasmic reticulum; N—nucleus; cm—cell membrane. Mag. 12,500X.

tron micrographs of the developing periodontal ligament. Figure 7 shows many nuclei and in the areas where nuclei are absent, cytoplasmic components are visible.

The cells in the region of the developing cementum are characterized by their abundant and vesicular endoplasmic reticulum. Between the walls of the endoplasmic reticulum a homogenous or granular substance fills the sac-like enlargements. This substance is seen to be more dense than the surrounding cytoplasm. In Figures 8 and 9, the walls of the endoplasmic reticulum can be traced from their narrowest point to an area where the walls are widely separated. Palade and Porter[8] termed these enlargements, cisternae. They are visualized as three-dimensional structures, broad in one plane and narrow in another.

Mitochondria are seen in close proximity to the endoplasmic reticulum. Granules of varying sizes are observed scattered within the cytoplasm. Dense granules are seen associated with the endoplasmic reticulum. These granules also seem to be "attached" to the cytoplasmic surface of the membrane.

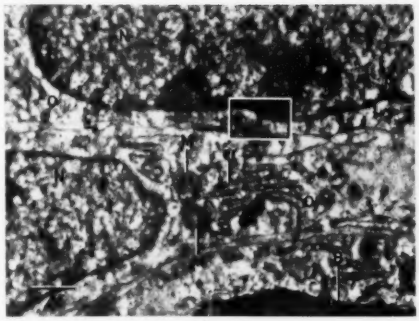

Fig. 4. Two osteoblasts (O) with their nuclei (N) and endoplasmic reticulum (er). Mitochondria (M) are seen in close proximity to the endoplasmic reticulum. B—grid. Mag. 12,500X.

The nucleus of the cell has a paired membrane (Figure 10) and the nuclear material is collected in clumps along the membrane.

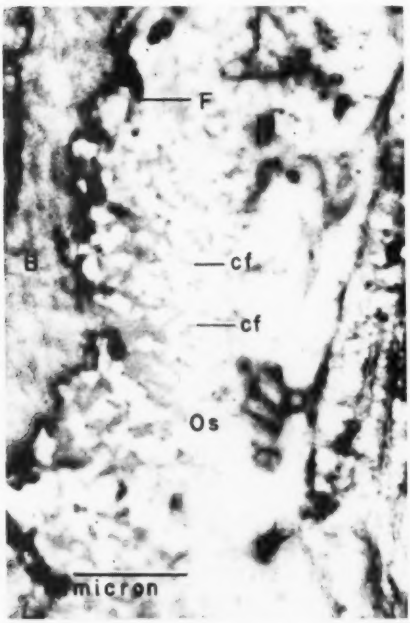

Fig. 3. High magnification of a section showing the fibers (cf) in the osteoid (os). The bone (B) also has fibers (F) projecting into the osteoid. Mag. 21,700X.

DISCUSSION AND REVIEW OF THE LITERATURE

Many investigators observed that osteoblasts arrange themselves around developing spicules of bone. In electron micrographs it appears that the osteoblasts seem to form a syncytium around developing bone.[5, 10, 11]

Jackson,[12, 13] Scott and Pease[14] and Sheldon and Robinson[11] described osteoblasts. Electron micrographs show that the cytoplasm contains endoplasmic reticulum with associated small dense granules. Mitochondria and dense granules of varying sizes are also identified within the cytoplasm of osteoblasts.

The osteocyte seen in Figure 6 indicates that the immature bone cell completely fills its lacuna.

The outer nuclear membrane of certain cells is known to be continuous with the "profiles" or walls of the endoplasmic

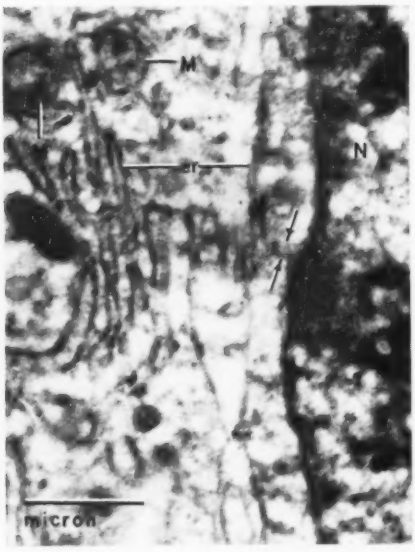

Fig. 5. Osteoblast enlarged from Figure 4. The endoplasmic reticulum (er) and mitochondria (M) are seen in the cytoplasm. Arrows indicate the connection between the endoplasmic reticulum and the nuclear (N) membrane. Mag. 25,000X.

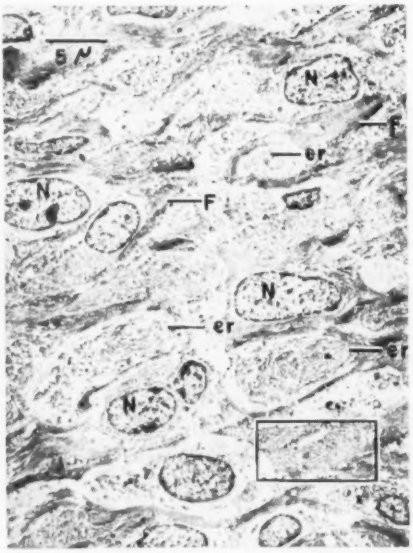

Fig. 7. Micrograph of the developing periodontal ligament near the root of the rat molar. Many nuclei (N) are present and where they are absent the endoplasmic reticulum (er) of the cell can be observed. Fiber (F) bundles are present between the cells. Mag. 2,500X.

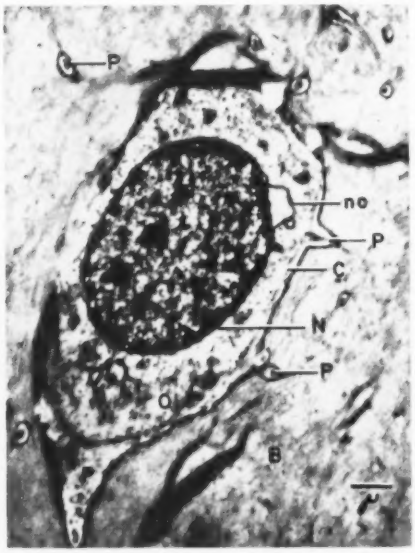

Fig. 6. Section of bone (B) showing an osteocyte (O) within its lacuna. Note the nuclear membrane (nc) projecting into the cytoplasm. N—nucleus; C—cell membrane; P—protoplasmic process. Mag. 8,200X.

reticulum. Watson[9] and Porter[15] have shown such connections in cells from the rat spleen and notocord of the Amblystoma larva. The osteoblast seen in Figure 5 also shows such connection of the nuclear membrane and endoplasmic reticulum. The dense homogenous substance within the endoplasmic reticulum is seen to be confluent with the perinuclear space. A distinction is visible between homogenous substance and the nuclear material. The space between the two nuclear membranes is termed the perinuclear space.[9]

Electron micrographs of the developing periodontal ligament show numerous cellular elements (Figure 7). Between the cells, fiber bundles are observed and their cut ends can be seen with high magnification (Figure 8).

Of particular interest is the endoplasmic reticulum within the cytoplasm of these cells. Palade and Porter[7] described the endoplasmic reticulum in fibroblasts of skin and heart as well as glandular epithelia. They

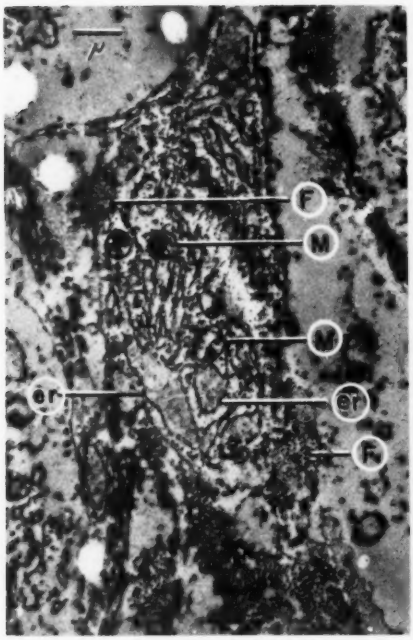

Fig. 8. Enlargement of boxed area in Figure 7 showing a vesicular type of endoplasmic reticulum (er) and mitochondria (M). A granular substance appears to fill the sac-like cavities of the endoplasmic reticulum. Cut ends of fiber (F) bundles are observed in the vicinity of the cell. Mag. 8,000X.

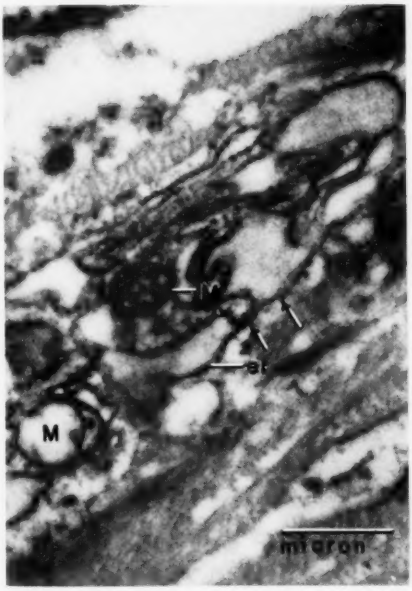

Fig. 9. High magnification of the endoplasmic reticulum from a cell of the developing periodontal ligament. Note the narrow walls of the endoplasmic reticulum enlarge to form sac-like cavities (arrows). M—mitochondrion; er—endoplasmic reticulum; P—homogenous substance within the endoplasmic reticulum. Mag. 21,700X.

stated that they were "relatively large, flattened vesicles of irregular outline." These flattened vesicles of the endoplasmic reticulum were termed cisternae.

The cells of the developing periodontal ligament in Figure 7 show abundant cisternae. Small dense granules appear to be "attached" to the cytoplasmic surface of the endoplasmic reticulum. Palade[8] first described and named them "small particulate granules." They are now more commonly known as Palade's granules. The granules were present in more than 40 different cell types. They appear in large numbers in embryonic cells, basal layer of epidermal cells and are especially numerous in glandular cells. Palade and Siekevitz[16, 17] provided evidence that these granules are ribonucleoprotein in nature and that they are very likely responsible for basophilia. Clermont[18] established, histochemically, that in rat spermatids these small granules alone are responsible for basophilia.

Studies with fractionated liver cells by ultracentrifugation separated small granules (100-250 A. in diameter). These granules were insoluble in sodium deoxycholate solution and contain almost 50% ribonucleic acid (RNA).[19]

That this system of vesicles and granules contains the major portion of cytoplasmic RNA is well established. Haguenau[20] also considers this system to be the site of protein synthesis. Brachet[21] supports this view by stating a high RNA concentration is more important in protein synthesis than the energy producing systems present in mitochondria.

Investigations established then that actively secreting cells have an highly organized endoplasmic reticulum and that cells not involved in protein synthesis have a small amount of endoplasmic reticulum.

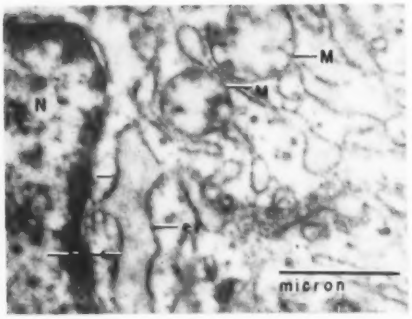

Fig. 10. Cell from the developing periodontal ligament showing paired nuclear membrane (arrows). N—nucleus; M—mitochondrion; er—endoplasmic reticulum; P—Palade's granule. Mag. 28,700X.

Porter and Pappas[22] described in vitro fibroblasts from chick embryo dermis in their study of fibrogenesis. The structure of the endoplasmic reticulum of these fibroblasts closely resembled the endoplasmic reticulum of the cells of the developing periodontal ligament.

SUMMARY AND CONCLUSIONS

Undecalcified thin sections of the investing tissues of the teeth have been studied with the electron microscope. The high magnification and improved resolution will provide additional knowledge of the submicroscopic structure of these tissues.

The presence of the organized endoplasmic reticulum in cells of the developing periodontal ligament has been revealed by electron microscopy. Because this cytoplasmic feature is characteristic of cells engaged in protein synthesis, it is suggested that these cells may produce a substance for the formation of fibers of the periodontal ligament.

The immature osteocyte fills its lacuna and protoplasmic processes are evident within the canaliculi.

Further investigation with the electron microscope may increase our present understanding of the developing and maturation of the periodontium.

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Methods and Values of Tooth Planing in Periodontal Therapy

by RICHARD CHACE, D.D.S., *Orlando, Florida*

AT the Centennial Congress of the Swedish Dental Society last summer Hilding Bjorn of the University of Malmo, presented a paper on the prevention of periodontal disease, including this paragraph.

> The direct cause of progressive periodontitis is the accumulation on the tooth surface of bacteria, which are bound together by a viscous mucoid mass to form a plaque. Active products diffuse into the periodontium and give rise to the first inflammatory changes, among them being an exudation of tissue fluid. This provides the microflora with a constantly renewed substrate, which permits further growth of the bacterial population; and this, in turn, leads to more pronounced inflammation, increase in exudate, and so the process continues. A vicious circle is set up with the bacterial plaque as its center.

The concept that bacteria play an important role in periodontal disease is certainly not a new one. It has been generally believed however, that since bacteria are always present in the mouth, calculus and rough margins of fillings are the more important etiological factors. These are generally considered to be the direct irritants and bacterial invasion is usually possible following this irritation. Recently Waerhaug[1] has attacked this thesis. He feels that the irritating qualities of calculus and rough fillings are chiefly due to the accumulation of bacterial plaques on their surfaces. This accumulation has been repeatedly shown by many investigators. Arnim[2] has also stressed the importance of plaque presence in periodontal disease and has revived an effective means of demonstrating their presence clinically by the use of a disclosing stain. The role of the plaque in calculus formation has, like its inherent pathogenicity, not been proven. Most authorities agree, however, that it is very likely an important factor.

If one can accept the premise that the bacterial plaque is a prime etiological factor in the production of periodontal disease, or that it is of importance in the formation of calculus, the need of its daily removal by the patient must be acknowledged. In a normal mouth the plaque formation is well controlled by tooth brushing. In a periodontally involved mouth plaque formation seems more tenacious. Tooth smoothness is an important factor. Swartz and Phillips[3] have shown in invitro studies that bacteria accumulate to a greater degree per unit of time on a rough surface than on a smooth surface. The bacteria are retained on such rough surfaces, even after vigorous brushing. This would explain the common clinical observation that an unglazed porcelain pontic produces irritation of the immovable tissue of a ridge, while highly

glazed porcelain does not. Years ago G. V. Black[4] showed that calculus would not form on a perfectly smooth surface.

If the cervical enamel, the cemento-enamel junction, and the exposed root of the tooth were smooth, there would be no need of doing more in the treatment of a pocket than removing the soft tissue wall, contouring the supporting tissues, and removing calculus. The patient would, even in a non-detergent mouth, be able to remove the constantly recurring plaque with his normal home care efforts. It is our observation, however, that the tooth itself presents varying degrees of roughness and softening. It is reasonable to interpret this softening as the hard tissue lesion produced by the plaque. Strangely enough it has not been described by the histopathologists even though its presence can be demonstrated by a sharp instrument. Sometimes the lesion is very superficial and is removed by a few strokes of a scaler or curet. More often it penetrates a considerable distance into the tooth. Riffle[5] has shown that this softening is deeper at the cemento-enamel junction, as would be expected, since it is at this point that the plaque has been on the tooth for the longest time. This lesion favors the re-formation of the plaque and it interferes with the plaque's removal by home care procedures.

Root planing is the operative procedure that removes this affected tooth structure. Its object is the production of a smooth, highly glazed surface on which bacterial plaques form with difficulty and from which plaques can easily be removed by brushing.

Root planing, under various names, is the oldest periodontal therapy. Most of the pioneer periodontists laid great stress on removing necrotic cementum and remnants of the detached periodontal membrane as well as the removal of calculus. The term "ivory-like smoothness" is often encountered in the literature. Generally it was felt that the cementum should not be removed in its entirety. Schaffer's recent work on root planing, however, seems to prove that it is impossible to smooth roots without removing most, if not all, of the cementum. Zander's[7] description of calculus penetration deep into cemental pits shows that simple scaling cannot ever remove all calculus. The descriptions of subgingival curettage encountered in the literature leads one to suspect that the thoroughness of early technics removed more tooth structure than the operators realized. The observations made while watching several modern conservative periodontists confirms this suspicion. Old treated cases that have been successful over a long period of time often show considerable reduction in cervical areas of the teeth. Riffle[8] treated his cases by root planing alone, not even giving his patients tooth brush instruction. He was able to get good results by carrying planing to its ultimate—that is, the rendering of each surface of each tooth glassy smooth.

The development of the various techniques of gingivectomy has tended to ignore root planing, although Kirkland[9] laid stress on root smoothing in describing his operation. The description of the various surgical procedures found in most modern texts scarcely mention the hard tissue wall of the pocket other than to urge pre-surgical scaling. It is only in describing reattachment procedures that root planing is advocated. It must be admitted, however, that in thorough scaling some planing is done.

The only way of evaluating root planing in periodontal therapy is to remember that it is only one phase of treatment and that its importance varies not only with the case, but with the skill and diligence other phases of treatment are executed. Here are some of the variables present. First, some periodontal cases respond well, even if they are incompletely treated. Secondly, an extremely cooperative patient might be successful in keeping his teeth free of plaques even though the root surfaces are far from smooth. Third, the careful contouring of the soft tissues in the various surgical procedures certainly makes it easier for the patient to remove plaques. On the other

hand ideal root smoothness is particularly important when it is impossible to completely eliminate the pathological crevice or to get good gingival architecture. Fourth if the occlusion constitutes the major problem it is illogical to expect either planing or surgery to be very effective.

There is danger in expecting too much of root planing in purely conservative procedure since it is often impossible to provide proper architecture and a shallow crevice by planing alone. In like manner, the operator can depend too much on surgery of the supporting tissues and can be misled by the apparent success of such measures. Pockets tend to recur by the same processes that produced them originally. It must be remembered that the plaque forms and is attached to the tooth. The high percentage of failure in periodontal therapy should cause each operator to appraise his efforts carefully and to be sure he is using every means at his disposal in the treatment schedule.

PRELIMINARY SCALING

In general it is well to precede root planing with the removal of gross deposits of calculus and the teaching of home care procedures. This preliminary root scaling or spot curettage may be done with any of the conventional instruments. Since considerable force must often be used, it is well that the instruments be rather dull so that there will be less danger of grooving or nicking the root surface.

Root planing is curettage performed with root smoothness as its objective rather than calculus removal. The operator has no way of knowing how much affected tooth structure must be removed before a hard, glassy surface can be obtained. A few strokes may accomplish this aim on one surface, whereas many minutes may be required on the next one.

Although any type of instrument can be employed, very sharp curets and Towner planes have been most effective in my experience. Planing can be done either by itself, with soft tissue curettage, or with the surgical removal of the soft tissue wall of the pocket. In any case the hard tissue wall is treated exactly the same way. If planing alone is done, only a very few teeth are treated at each appointment. If quadrant soft tissue surgery is done, it is sometimes impossible to completely plane so large an area at the time of the operation because of the limitations of time and anesthesia. Further planing must be done at the pack change and the pack removal.

As large an instrument as possible should be used in planing, especially in the readily accessible region of the cemento-enamel junction. Since there is usually more softness or roughness in this area, the large instruments will remove more rapidy the affected tooth structure without producing grooves. When the soft tissue is removed, the larger curets can be used for most of the planing. Particular attention must be given to the concavities present on the tooth surfaces. Beginning bifurcation involvements present grooves that must be converted to easily cleaned depressions. In some areas rotary instruments may be helpful in the operation. Fine cuttlefish strips can often be used interproximally.

When the soft tissue wall is not removed, it is necessary to use finer instruments in the deeper areas so that the sense of touch is not impaired by the binding of the soft tissue wall on the back of the instrument. The finer the instrument, the lighter and longer the stroke. As many different shapes and types of instruments as possible should be used on each surface; and each surface should be planed in as many directions as access will allow. As the tooth becomes smoother the instrument should be held more lightly. Resharpening must be continuous during the operation. It must be emphasized that the sense of touch is more important than the sense of sight. It is almost impossible to see the root surface well enough to judge its smoothness.

The operator should be encouraged but not misled by improvement in the feel of

the tooth. It is very common to accept a result that is less than adequate. A stethoscope with the bell removed is a useful tool in teaching root planing. The handle of a sharp curet is inserted in the rubber tube and the difference in sound between the smooth and the fairly smooth can be readily distinguished. Sometimes the goal of smoothness cannot be attained without severe tooth mutilation. Complete elimination of the pathological crevice and the attaining of physiological contours of the supporting tissues becomes of even more importance when any roughness must be left.

Occasionally the lesion of the roots can reach such a depth that it is best to restore the tooth with an inlay or crown. On the other hand, planing can often remove beginning decay, particularly Class V cavities, and can make unnecessary deep gingival extension of proximal preparations. A hard, smooth tooth surface is an ideal one on which to finish cavity margins. We have all seen re-decay around the margins of inlays and crowns because the dentist could never find a good place to end his preparations. Root planing before restoration would make this failure less common.

It is important to have some means of determining whether plaque control has been established in each periodontal case. A practical way of accomplishing this is not to dismiss the patient until several weeks after the pockets are eliminated and healing has occurred. If examination at this time reveals excessive plaque or subgingival calculus formation in spite of reasonably good home care, re-planing can be done. Cooperation is expected; a patient using a nondetergent diet must be given almost perfect root smoothness and gingival contour. Root smoothness reduces the care necessary in the maintenance of any periodontal patient. Less calculus forms between visits, and that which does form, is more easily removed. Many operators have noted that their treated cases require less and less supervision as time goes on. This is because the repeated scalings have, often inadvertently, removed the softened tooth structure and have reached the smooth, self cleansing surfaces. This improvement occurs even though the patient admits a reduction in his home care efforts. Sometimes a case will get along well generally but will reveal a single area of inflammation at a recall appointment. Examination will show a slight roughness of the tooth surface next to the area. The thorough re-planing of this surface will correct the condition.

SUMMARY

1. The bacterial plaque attached to the tooth must be regarded as an important factor in periodontal disease.

2. The easy removal of plaques by the patient's home care efforts is of prime importance in the prevention of the recurrence of pockets.

3. Smooth tooth surfaces as well as minimal crevice depth and good gingival architecture are major objectives of treatment.

4. Root planing, or the smoothing of the hard tissue wall of the pocket, enables this objective to be achieved.

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The Periodontal Status of Boys 11 to 17 Years Old in Bombay, India

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THIS investigation had the following objectives:

1. To train investigators for epidemiological studies of periodontal disease in India;

2. To initiate a study of the prevalence and incidence of periodontal disease, with special reference to early destructive periodontal disease;

3. To relate various etiologic factors to the prevalence and severity of the periodontal disease;

4. To investigate whether or not periodontal disease in India is of a different nature or type than periodontal disease in the Western world;

5. To gain information that could serve as a basis for planned management and prevention of periodontal disease.

A review of the literature pertinent to the planning of this study has been published previously.[1]

MATERIAL AND METHOD

Since one of the main purposes of this investigation was to study the initial phase of destructive periodontal disease in the permanent dentition, it was decided to examine age groups between 11 and 17 years. According to local authorities in Bombay, it was more expedient to select boys for the survey than girls since the boys were attending school more regularly than girls. By studying only boys the problem of possible differences in periodontal scores associated with the difference in sex if mixed groups were examined could be avoided.

An attempt was made to examine urban and rural groups of equal size and comparable age. The selected population groups were all living under very poor economic conditions. Urban public schools in the cotton mill district of Bombay provided the urban sample. The rural population groups were made up of boys from public and missionary schools in the Bassein Road area, about 40 to 50 miles from the center of Bombay. This area is an agricultural district (rice and bananas) but, being situated so close to the city of Bombay, the economic and social standards cannot be accepted as typical of rural India.

In the 11-year group were included boys who were 11 years of age or older but had not reached the age of 12 at the date of the examination. The 13, 15, and 17-year old groups were formed in the same way. All of the boys within these age groups were examined in every school visited. The cooperation from the school authorities, teachers, and children was excellent. The examination usually was done on open balconies without artificial light, and with instruments described in a previous article.[1]

Eight Indian dentists participated in the examination procedures. Each boy within the selected age group was examined twice in order to complete a Russell-Greene scoring under Dr. Greene's supervision[2] and another scoring according to the system

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This investigation was sponsored jointly by the World Health Organization and the government of India during the summer of 1957. A detailed report was submitted to W.H.O. in December, 1958. A large number of persons participated in the organization and execution of this study, but space will allow acknowledgment only to a few who especially devoted time, interest, and untiring effort towards the completion of the project: From W.H.O., Dr. C. L. Sebelius; from India, Colonel N. N. Bery, Drs. H. D. Merchant, K. L. Shourie, M. K. Basu, R. P. Dastur, N. T. Desai, B. R. Kankaria, (Miss) S. V. Koppikar, (Miss) M. A. Malaowalla, S. B. Sathe, S. B. Vaid; from the U. S. Public Health Service, Dr. J. C. Greene.

TABLE II
Periodontal Disease, Calculus, Plaque, and Attrition Scores

	Age	Number Examined	Mean Scores			
			Periodontal Disease	Calculus	Plaque	Attrition
Bombay City	11	343	1.33 ± 0.026*	0.91 ± 0.030*	1.73 ± 0.029*	0.40 ± 0.018*
	13	255	1.45 0.029	1.20 0.034	1.65 0.031	0.56 0.021
	15	189	1.53 0.033	1.23 0.043	1.54 0.035	0.61 0.023
	17	40	1.42 0.065	1.33 0.074	1.55 0.090	.75 0.052
Urban Total	11–17	827	1.42 ± 0.016	1.09 ± 0.019	1.65 ± 0.018	0.51 ± 0.011
Bassein Road	11	202	1.55 ± 0.032*	1.13 ± 0.036*	1.96 ± 0.035*	0.69 ± 0.021*
	13	209	1.56 0.033	1.30 0.033	1.88 0.027	0.77 0.018
	15	203	1.55 0.034	1.40 0.033	1.75 0.031	0.88 0.016
	17	177	1.61 0.040	1.53 0.048	1.67 0.033	0.95 0.016
Rural Total	11–17	791	1.57 ± 0.017	1.33 ± 0.019	1.82 ± 0.016	0.82 ± 0.009
Rural	19–22	19	1.97 ± 0.129*	1.69 ± 0.090*	1.52 ± 0.135*	1.09 ± 0.044*
	23–26	27	2.13 0.126	1.72 0.071	1.60 0.059	1.27 0.048
	27–30	13	2.43 0.254	1.83 0.101	1.72 0.065	1.29 0.052
Total		59	2.14 ± 0.030	1.73 ± 0.049	1.60 ± 0.053	1.22 ± 0.029

*Standard Error

suggested by Ramfjord.[1] An attempt was made to familiarize the examiners with both systems and to let them practice each scoring system.

The findings obtained according to the Russell-Greene system have been published by Dr. Greene.[2] His article is referred to for comparison with the findings reported here following the use of the periodontal disease index (P.D.I.) described by Ramfjord.[1]

The main survey included 827 boys from Bombay City and 791 boys from the Bassein Road district. In addition, 59 males, 19 to 30 years old, were examined at a teacher's college in the Bassein Road district.

FINDINGS

Table I is a compilation of all recordings with regard to periodontal disease, calculus, plaque, and attrition.

A trend for periodontal disease to increase slowly in severity with increasing age is apparent from this table. An exception to this trend seems to be the 17-year old boys in the urban section. There also appears to be a statistically significant difference at the one per cent level between the severity of periodontal disease in the rural and urban populations for most age groups with the rural groups having the highest scores. A similar difference also was reported by Greene.[2] However, some modifying factors should be considered in order to understand and evaluate this difference.

1. The urban examinations were done first. The examiners were not well trained, and the ability to observe periodontal disease increases with the experience of the examiner.

2. Three examiners from the urban study did not participate in the rural study. One examiner, whose scores were consistently far below average in the urban study, was used as interpreter and thus practically eliminated from examination of subjects in the rural study (examined only 6 boys).

In order to minimize the examiner difference, Table II was compiled. Only the findings of three examiners (including the

TABLE II
Periodontal Disease, Calculus, Plaque and Attrition Scores by the Three Most Consistent Examiners

	Age	*Number Examined*	*Mean Scores*			
			Periodontal Disease	*Calculus*	*Plaque*	*Attrition*
Bombay City	11	82	1.72 ± 0.036*	1.09 ± 0.057*	1.84 ± 0.055*	0.579 ± 0.036*
	13	93	1.73 0.036	1.27 0.050	1.72 0.051	0.695 0.035
	15	76	1.65 0.043	1.35 0.056	1.54 0.046	0.746 0.022
	17	13	1.56 0.077	1.45 0.092	1.56 0.067	0.869 0.061
Urban Total	11–17	264	1.70 ± 0.021	1.25 ± 0.030	1.70 ± 0.028	0.682 ± 0.018
Bassein Road	11	141	1.69 ± 0.029*	1.05 ± 0.037*	1.97 ± 0.039*	0.699 ± 0.020*
	13	140	1.74 0.030	1.25 0.041	1.87 0.034	0.769 0.020
	15	138	1.72 0.034	1.37 0.042	1.75 0.040	0.872 0.019
	17	121	1.71 0.045	1.46 0.038	1.64 0.041	0.946 0.018
Rural Total	11–17	540	1.72 ± 0.017	1.28 ± 0.020	1.81 ± 0.019	0.817 ± 0.010

*Standard Error

writer) whose scores were very similar have been included. According to this table there is only a slight but non-significant difference between the periodontal disease scores of the urban and rural boys.

A gradual increase in accumulation of calculus with age is evident in both Tables I and II. However, the statistically significant difference (at the one per cent level of confidence) in Table I between the rural and urban groups appears as an insignificant difference in Table II, indicating only slightly more calculus in the rural than in the urban group.

The scores for stainable dental plaques gradually decline with an increase in age from 11 to 15 years in the urban boys and from 11 to 17 years of age in the rural boys according to Tables I and II. The plaque scores are also significantly higher in the rural than in the urban groups (some at the 5 per cent level and some at the one per cent level).

A gradual increase in attrition with age is obvious from both Tables I and II. There is also a highly significant difference in attrition between the rural and urban groups of the same age. The rural groups consistently exhibit more occlusal and incisal wear than the urban groups. No attempt was made to study the possible dietary background for the higher attrition scores in the rural groups.

At the bottom of Table I are listed the scores from a pilot study of men 19 to 30 years of age. This limited material was divided in age groups of four-year intervals: 19 through 22 years, 23 through 26, and 27 through 30 years of age. From this table, it appears that periodontal disease slowly increases in severity (amount of periodontal destruction) with increase in age. A slow gradual increase also is evident for the calculus, plaque, and attrition scores. It is interesting to note that the level of plaque appears to reach a low at 15 to 19 years of age, seemingly somewhat earlier in the urban than in the rural population, and then the score gradually increases with age.

The entire material was analyzed with reference to prevalence of periodontal disease, periodontal pockets, and gingival recession (Table III). This table demonstrates drastically the magnitude of the problem of periodontal disease in India since practically 100 per cent of all individuals examined had periodontal disease. From this table, it also appears that periodontal pockets are initiated after the age of 15 years. The very few individuals who had true periodontal pockets or gingival

TABLE III
Persons with Periodontal Disease, Periodontal Pockets and Recession (Urban and Rural)

Age	Number Examined	Persons with Periodontal Disease		Persons with Periodontal Pockets		Persons with Recession	
		Number	Percent	Number	Percent	Number	Percent
11	542	539	99.4	4	0.7	3	0.6
13	464	464	100.0	3	0.6	1	0.2
15	392	392	100.0	8	2.0	4	1.0
17	217	217	100.0	20	9.2	7	3.2
19-22 (mean 20.6)	19	19	100.0	6	31.6	3	15.8
23-26 (mean 24.0)	27	27	100.0	13	48.1	7	25.9
27-30 (mean 28.3)	13	13	100.0	6	46.2	6	46.2

recession before the age of 15 had these anomalies in association with marked malposition of the teeth, and the epithelial attachment was situated only slightly apical to the cementum-enamel junction. At the age of 17, more than 9 per cent of the individuals examined had periodontal pockets, and in the small group of 19 to 22 year old individuals, 31.6 per cent had periodontal pockets. Much larger population groups have to be examined before significant results can be obtained relative to the progress of periodontal disease in individuals older than 17 years of age. However, it is interesting to note the great increase in gingival recession with age. From clinical observation, it was obvious that this recession was due to horizontal rubbing over the teeth with the fingers for the purpose of oral hygiene. This procedure was often accompanied by the use of sharp abrasives (ashes or abrasive tooth powders), producing wide "U" shaped notches on the labial gingival aspect of the teeth, especially in the maxilla in persons older than 25 to 30 years of age.

The gingivitis score for the individual teeth in the various age groups is recorded in Table IV. The recorded mandibular lower incisor (tooth #25) consistently showed the most severe gingivitis in its surrounding tissues. The next highest score for gingivitis was found around the maxillary first molar (tooth #3), while the lowest score for gingivitis was found around the mandibular first bicuspid (tooth #28). A slight decrease in the mean score for gingivitis from 15 to 17 years of age is consistent with the reports from other surveys of prevalence of periodontal disease.[3-7]

The distribution of the periodontal pockets is recorded in Table V-A. It is

TABLE IV
Gingivitis Score for Individual Teeth (Urban and Rural)

Age	Number Examined	Tooth #3	Tooth #9	Tooth #12	Tooth #19	Tooth #25	Tooth #28	Mean Gingivitis
11	542	1.48	1.27	1.30	1.38	1.69	1.09	1.40
13	464	1.62	1.40	1.47	1.51	1.81	1.13	1.49
15	392	1.60	1.43	1.46	1.55	1.87	1.27	1.53
17	217	1.63	1.39	1.50	1.50	1.89	1.25	1.50

TABLE V-A
Individual Teeth with Periodontal Pockets (Urban and Rural)

Age	*Number of Cases*	*Tooth #3*	*Tooth #9*	*Tooth #12*	*Tooth #19*	*Tooth #25*	*Tooth #28*	*Total Number of Teeth with Periodontitis*
11	542	0	1	0	0	3	0	4
13	464	0	0	1	0	2	0	3
15	392	0	0	2	1	5	0	8
17	217	8	2	4	4	9	1	28

evident that the greatest number of periodontal pockets were found around tooth #25 which, according to Table IV, also showed the highest score for gingivitis. Tooth #3, which had the next highest score for gingivitis, also had the next highest number of periodontal pockets. Tooth #28, which had the lowest score for gingivitis, had the lowest number of periodontal pockets. In the 17-year old group, periodontal pocket formation had been initiated around one or more of the teeth included in the examination in one or more individuals.

Gingival recession is recorded in Table V-B. The figures for recession were low in these young age groups, and the recession usually was found in association with severe malposition of the teeth and most often where the lower incisors were in extreme labial version.

Tables VI-A and VI-B respectively show the amount of calculus on the individual teeth and the percentage of teeth with calculus of all the teeth examined within the various groups. The mandibular lower incisor (tooth #25) is leading both in regard to amount of calculus and in percentage of teeth with calculus. The calculus deposition on tooth #3 shows the next highest figures, and the lowest score for calculus was found on tooth #28. Note the steady increase in amount of calculus and number of teeth with calculus with increase in age as evident from Tables VI-A and VI-B.

The distribution of bacterial dental plaques is recorded in Tables VII-A and VII-B. A slight decrease in the amount of plaque on the teeth with age can be observed from Table VII-A. This decrease is found mainly in the age groups from 11 to 15 years of age, and it was observed clinically to be associated with plaque removal on the labial aspect of the teeth by rubbing the teeth with the finger tip and some abrasive. On the other hand, it is evident from Table VII-B that the percentage of teeth with bacterial plaques increased with age until it involved 95 to 100 per cent of all of the teeth examined.

Table VIII shows the mean scores for attrition, mobility, and open contact for the entire material. A gradual increase in attrition with age is evident from this

TABLE V-B
Individual Teeth with Recession (Urban and Rural)

Age	*Number of Cases*	*Tooth #3*	*Tooth #9*	*Tooth #12*	*Tooth #19*	*Tooth #25*	*Tooth #28*	*Total Number of Teeth with Recession*
11	542	0	0	0	0	2	0	2
13	464	0	0	0	0	2	0	2
15	392	0	0	0	0	4	0	4
17	217	2	1	1	0	0	5	9

TABLE VI-A
Calculus Score for Individual Teeth (Urban and Rural)

Age	Number Examined	Tooth #3	Tooth #9	Tooth #12	Tooth #19	Tooth #25	Tooth #28	Mean Calculus
11	542	1.23	.90	.62	1.04	1.37	.43	.99
13	464	1.56	1.24	1.05	1.31	1.66	.63	1.25
15	392	1.50	1.29	1.17	1.45	1.62	.91	1.48
17	217	1.65	1.47	1.50	1.56	1.66	1.11	1.53

TABLE VI-B
Individual Teeth with Calculus (Urban and Rural)

Age	Number Examined	Tooth #3		Tooth #9		Tooth #12		Tooth #19		Tooth #25		Tooth #28	
		No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%
11	542	377	69.6	263	48.5	151	27.9	346	63.8	435	80.3	87	16.0
13	464	406	87.5	297	64.0	265	57.1	332	71.6	421	90.7	175	37.7
15	392	343	87.5	267	68.1	265	67.6	315	80.4	353	90.0	225	57.4
17	217	206	94.9	171	78.8	184	84.8	194	89.4	201	92.6	147	67.7

TABLE VII-A
Plaque Score for Individual Teeth (Urban and Rural)

Age	Number Examined	Tooth #3	Tooth #9	Tooth #12	Tooth #19	Tooth #25	Tooth #28	Mean Plaque
11	542	2.00	1.70	1.50	1.91	1.98	1.61	1.83
13	464	1.95	1.55	1.58	1.85	1.95	1.59	1.75
15	392	1.89	1.59	1.52	1.80	1.77	1.56	1.67
17	217	1.74	1.54	1.53	1.76	1.79	1.52	1.65

TABLE VII-B
Individual Teeth with Plaque (Urban and Rural)

Age	Number Examined	Tooth #3		Tooth #9		Tooth #12		Tooth #19		Tooth #25		Tooth #28	
		No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%
11	542	538	99.3	502	92.6	386	71.2	493	91.0	513	94.6	297	54.8
13	464	459	98.9	435	93.8	421	90.7	445	95.9	451	97.2	396	85.3
15	392	386	98.5	377	96.2	374	96.9	370	94.4	376	95.9	360	91.8
17	217	214	98.6	217	100.0	217	100.0	217	100.0	208	95.9	209	96.3

TABLE VIII
Mean Scores for Attrition, Mobility and Open Contact (Urban and Rural)

Age	*Number Examined*	*Attrition*	*Mobility*	*Contact*
11	542	.57	.02	.19
13	464	.65	.01	.17
15	392	.75	.01	.17
17	217	.92	.02	.19

table. There are no significant variations in mobility and open contacts in these young age groups. The percentage of the teeth examined that showed evidence of attrition is recorded in Table IX. The increase of attrition with age and its relation to the number of years that the teeth have been present in the mouth can be observed from this table.

From Table X, it is evident that tooth #25 exhibited abnormal mobility more often than the other teeth examined. About 5 per cent of the mandibular right central incisors showed a non-physiologic degree of mobility. From clinical observation, it was evident that this increased mobility was due to (1) obvious malposition and (2) severe periodontal inflammation. The figures for mobility for the rest of the teeth are insignificant in this material of young individuals.

Open contacts were observed most commonly around tooth #9 according to Table XI. About one-third to one-fourth of these teeth had open contacts. There was no distinct age distribution for the open contacts. Open contacts were observed much more rarely around the upper and lower first molars.

A cursory attempt was made to compare the methods of oral hygiene with the score for periodontal disease (Table XII), but the number of individuals using brush and datan stick was too small to serve as a basis for an evaluation of these various methods. Seemingly, the use of a brush was associated with a slightly lower periodontal disease score than the other methods of oral hygiene. It should be emphasized that the inadequate brushes used and the haphazardous way they were used do not in any way allow an evaluation of the value of tooth brushing in the prevention of periodontal disease.

The mean depth of the gingival crevice of all teeth examined (Table XIII) shows only a slight variation with age. A small decrease in crevice depth with increase in age is noted on the buccal and lingual side of the teeth, while the interproximal crevice depth is unaltered or slightly increased.

Table XIV shows the mean periodontal

TABLE IX
Individual Teeth with Attrition (Urban and Rural)

Age	*Number Examined*	*Tooth #3*		*Tooth #9*		*Tooth #12*		*Tooth #19*		*Tooth #25*		*Tooth #28*	
		No. of Teeth	*%*	*No. of Teeth*	*%*	*No. of Teeth*	*%*	*No. of Teeth*	*%*	*No. of Teeth*	*%*	*No. of Teeth*	*%*
11	542	324	59.8	334	61.6	89	16.4	348	64.2	255	47.0	31	5.7
13	464	359	77.4	342	73.7	167	36.0	383	82.5	299	64.4	103	22.2
15	392	318	81.1	333	84.9	198	50.5	325	82.9	297	75.8	138	35.2
17	217	200	92.2	197	90.8	152	70.0	179	82.5	195	89.9	118	54.4

TABLE X
Individual Teeth with Mobility (Urban and Rural)

Age	Number Examined	Tooth #3		Tooth #9		Tooth #12		Tooth #19		Tooth #25		Tooth #28	
		No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%
11	542	1	.18	2	.37	0	0	3	.55	38	7.0	0	0
13	464	2	.43	1	.22	0	0	4	.86	19	4.0	2	.43
15	392	0	0	3	.77	0	0	0	0	20	5.1	2	.51
17	217	0	0	1	.46	2	.92	1	.46	11	5.0	0	0

disease index (P.D.I.) for the various groups examined indicating a slow, gradual increase in severity of periodontal disease with age.

The caries problem in the permanent teeth seemed negligible in our material, while remaining deciduous teeth usually were totally broken down by caries as reported by Chaudhry et al[8] from another area of India. Some cases of arrested caries were observed in the occlusal surfaces of the first molars. Occlusal wear in these areas of arrested caries gave the appearance of deep and wide occlusal fissures with a rounded bottom, almost as though the fissure had been deepened with a #8 round bur.

No attempt was made to investigate the occurrence of caries, but from casual inquiry, it appeared that babies and small children often drank a heavy sugar solution. Later, when they entered school, this was considered "baby stuff" and its use was discontinued.

SIGNIFICANCE OF FINDINGS

The results should be evaluated in relation to the five objectives listed in the introduction.

1. The limited time and restricted facilities available did not allow for satisfactory training and calibration of the examiners assigned to this study. The relatively few subjects examined by some of the examiners and the simultaneous use of two similar, but in some respects different, scoring methods also detracted from the reliability of the collected data. An attempt was made to analyze statistically the comparability of the examiners, but too few subjects had been examined by the individual examiner to make this analysis meaningful. (A limited number of copies of the detailed report submitted to the W.H.O. is available upon request to the author.) A very close comparability of scores existed among the three to four examiners who were given the most instruction and supervision. Therefore, it can be

TABLE XI
Individual Teeth with Open Contacts (Urban and Rural)

Age	Number Examined	Tooth #3		Tooth #9		Tooth #12		Tooth #19		Tooth #25		Tooth #28	
		No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%
11	542	15	2.8	145	26.8	73	13.5	24	4.4	46	8.5	54	10.0
13	464	23	5.0	133	28.7	72	15.5	18	3.4	51	11.0	73	15.7
15	392	13	3.3	103	26.3	65	16.6	13	3.3	69	17.6	64	16.3
17	217	3	1.4	72	33.2	31	14.3	10	4.6	38	17.5	35	16.1

TABLE XII
Method of Oral Hygiene Related to Periodontal Disease (Urban and Rural)

Age	*Method of Cleaning*	*Number Examined*	*Mean Periodontal Disease Score*
11	Brush plus powder or paste	13	1.18
	Finger plus abrasive	514	1.40
	Stick	15	1.72
13	Brush plus powder or paste	17	1.56
	Finger plus abrasive	388	1.49
	Stick	15	1.43
15	Brush plus powder or paste	33	1.48
	Finger plus abrasive	371	1.70
	Stick	25	1.58
17	Brush plus powder or paste	31	1.44
	Finger plus abrasive	165	1.58
	Stick	25	1.52

concluded that the method would provide good comparability of examiners following adequate training.

2. Epidemiology of periodontal disease in India has been more thoroughly investigated than in most parts of the world. Marshall-Day and Shourie's pioneer surveys[9-12] have been followed by very fine investigations by Mehta, Sanjana, and others.[13-16] These studies, supported by the present report, have established a prevalence rate of periodontal disease so close to 100 per cent that further prevalence studies in similar population groups apparently will be of very limited value. However, the available information on incidence, severity, and quantitation of periodontal destruction is highly incomplete and controversial. The main emphasis in the present study, therefore, was directed toward an accurate clinical registration of the periodontal status in the subjects that were examined. The main shortcoming with all of the previously used recording systems for periodontal disease is the lack of orientation of the periodontal pockets that makes attempts to quantitate periodontal destruction unreliable or meaningless. The cementum-enamel junction provides a natural landmark for recording of periodontal pockets,[1] and only when this landmark is used can it be ascertained clinically whether or not periodontal support has been lost. Controlled conditions for such recordings requires suitable instruments and well-trained investigators in order to achieve results in clinical research that in reliability and significance can match the fine laboratory investigations in current dental research.

By far the most important finding in the present study was that the clinically measurable destruction of periodontal attachment to the tooth started around the age of 15 years. True periodontal pockets or gingival recession apically to the cementum-enamel junction were observed in more than 12 per cent of the boys at the age of 17 years (Table III), while 3 per cent had only an insignificant early loss of periodontal support at the age of 15 years. Obviously, this means that periodontal treatment should be initiated before the age of 15 years in order to safeguard against permanent loss of periodontal support under the conditions of the present study.

The figures for periodontal pockets and gingival recession (Table III, bottom) in subjects between 19 and 30 years of age show that 42.4 per cent had true periodontal pockets and 27 per cent had gingival recession apically to the cementum-enamel junction, indicating that 70 per cent of the subjects in this group had experienced permanent loss of periodontal support.

TABLE XIII
Mean Crevice Depth of All Teeth Examined

Age	Number Examined	Mesial	Distal	Buccal	Lingual
11	542	2.69	2.53	2.04	1.92
13	464	2.75	2.61	1.94	1.86
15	392	2.71	2.63	1.82	1.84
17	217	2.76	2.63	1.71	1.75

These figures are considerably higher than the figures for pocket formation reported by Greene[2] for the same group. This difference is probably due to a routine probing with a fine probe in the present study compared with probing with a scaler in areas where the presence of pockets is strongly suspected in compliance with the Russell method[17] used by Greene.[2]

The noted slight decrease in severity of gingivitis from 13 or 15 years of age to 17 years was counterbalanced in the P.D.I. scores (Table XIV) by the onset of pocket formation and gingival recession in these groups indicating a slowly progressing destruction associated with chronic periodontal disease in spite of a light improvement of the gingival inflammation.

The "statistically significant" difference in P.D.I. between the rural and urban groups in Table I has already been discussed. A warning should be made against implying too much significance to small so-called "statistically significant" variations in surveys of this type since the mathematical signifiance according to statistical methods may be clinically insignificant. Furthermore, statistics should be applied with great caution to clinical material where the basic data are dependent upon the ability of the examiner to observe, assess, and record rather subtile clinical variations.

3. Several factors that generally are considered important in the etiology of periodontal disease were studied.

The tremendous accumulation of calculus in these young individuals and the practically non-existent oral hygiene dominated completely over the other sources of gingival irritation. The gradual increase in amount of calculus and number of teeth with calculus with age is demonstrated in Tables VI-A and VI-B. By comparing calculus scores from Table VI-A with gingivitis scores for the same teeth in Table IV an obvious correlation appears between these two conditions. Even more convincing is the relationship between teeth with pockets (Table V-A) and the calculus scores for the same teeth (Table VI-A). A cause and effect relationship between calculus and periodontal disease has been reported by practically every investigator in this field since King's study[19] in 1945.

No explanation appeared for the severe and early calculus formation noted in the present and other investigations from Asia[2, 3, 5, 9-12, 16, 20, 21] and North Africa.[22] Neither the physical properties of the diet nor the nutritional values of the food were studied. However, in future studies of calculus formation in similar populations the diet should be given due attention. Chewing of betel leaves had nothing to do with

TABLE XIV
Periodontal Disease Index for Urban and Rural Groups Combined

Age	Number Examined	Total Periodontal Index
11	542	1.40
13	464	1.50
15	392	1.54
17	217	1.56

the calculus formation reported in the present study, since very few of these young boys indulged in that habit.

The calculus was soft and easily removed in the 11 and 13 year old boys; but in the 17 year old group, the calculus was much harder and more difficult to remove. The ease by which the calculus can be removed before 15 years of age and the onset of pocket formation at this age are both factors that should encourage the calculus removal before the age of 15 years.

No apparent correlation was found between the gingivitis and plaque scores for the individual teeth (Tables IV and VII-A). However, an indefinite correlation appeared between the gingivitis score (Table IV) and the percentage of teeth with bacterial plaques (Table VII-B). Chawla and coworkers,[23] using the scoring methods that were used in the present study for an age group from 12 to 30 years, found a progressive increase in the number of teeth that had the highest gingivitis score as the plaque score increased. The same trend can be found in Table I when the age group from 19 to 30 years of age is considered; however, multiple correlation tests with other etiologic factors were not done.

It is evident from clinical observation that the gingival part of the coronal plaque is the main source of the gingival irritation, and how much of the rest of the crown that is covered in very extensive plaque formation is apparently of lesser importance.

The present investigation of these young boys with early periodontal disease did not provide a suitable material for study of a possible relationship between attrition, mobility, open contacts, and the progress of periodontal disease.

4. All periodontal disease observed was clearly of inflammatory nature (gingivitis and periodontitis). In view of the overwhelming magnitude of the local irritants, especially calculus and bacterial plaques, and in spite of markedly retarded growth and apparent undernourishment among the majority of the examined subjects, the periodontal resistance to pocket formation must be considered surprisingly good in these young age groups.

Not a single case was observed that possibly could fit into the common clinical description of so-called "periodontosis." One or two individuals could be said to have "juvenile periodontitis," but they had considerable accumulation of calculus and severe inflammation. The only difference between these individuals and the rest of the subjects was in the degree of periodontal destruction rather than any difference in kind of disease.

5. The most significant finding related to management and prevention of periodontal disease in India was the determination of the age for onset of permanent periodontal destruction to about 15 years of age under the conditions of the present investigation. This observation has encouraged planning and initiation of an extensive program for oral prophylaxis in school children in India.[24] However, the main reason for the severe periodontal problem in India, the tremendous calculus formation, remains unexplained.

The findings with regard to methods for oral hygiene were inconclusive (Table XII) although the tendency was to confirm the observations of Chawla and coworkers[23] that datan sticks were better than the use of fingers, and toothbrushes were better than the use of datan sticks.

CONCLUSIONS

1. Almost 100 per cent of the individuals examined had periodontal disease.

2. True periodontal pockets did not occur to any significant extent before the age of 15 years.

3. The periodontal disease increased slowly in severity with increasing age, although gingival inflammation decreased slightly in the middle teens.

4. The amount of calculus increased with age.

5. The amount of bacterial plaques decreased from 11 to 15 or 17 years of age; later it increased.

6. A direct relationship was observed between the amount of calculus and the severity of periodontal disease.

7. The most severe gingival inflammation and pocket formation occurred around the lower incisors and the maxillary first molars. These teeth also had the highest calculus scores.

8. The lowest P.D.I. score was for the mandibular first bicuspid.

9. All periodontal disease was clearly of inflammatory nature (gingivitis and periodontitis).

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Clinical Evaluation of an Electrically Powered Toothbrush in Home Periodontal Therapy

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IT is generally agreed that proper dental hygiene must include the regular brushing of the teeth, and that such brushing should be performed with a vertical, rather than a horizontal, motion.[1-4] This is all the more true in the home care of patients who are receiving periodontal therapy.[1-3] The gingival massage provided by correct brushing should change the appearance of the gingivae of periodontal patients from soft, red, and boggy to firm, pink, and stippled. This change is a useful clinical index for the determination of the success of home care in periodontal patients.

Much study has been given to the technics of toothbrushing, the construction of toothbrushes and the relative advantages of various bristles and dentifrices.[5-7] Nevertheless, a conventional toothbrush of optimum design and construction is practically worthless, or even may be harmful, if improperly used.[9-11] Proper toothbrushing with the conventional toothbrush is not, of course, a difficult technic to master, but far too many patients are insufficiently motivated to do so.

Recently, an electrically operated toothbrush has been introduced that is designed to clean the teeth thoroughly without abrasion of the soft oral tissues and to perform a thorough gingival massage. To test the effectiveness of the instrument, it was decided to perform a home-use test of it with patients undergoing periodontal therapy.

MATERIALS AND METHODS

The Instrument. The electrically operated toothbrush (Broxodent*) consists, basically, of a miniature electric motor sealed into a plastic tube that serves as a handle. The seal is entirely water-tight, so there is no electrical hazard. From the lower end of this handle the power cord runs through a water-tight gasket; on the upper end is a stud upon which is placed the brush head, which is slotted to receive it. The motor is activated when the cord is plugged into an electrical outlet, but the oscillation of the brush head can be stopped by light finger pressure on either or both of two brake buttons, one on either side of the handle (Figure 1).

The motor oscillates the brush head rapidly through 60° of vertical arc, thus providing the correct up-and-down motion for toothbrushing and gingival massage. The brush head is small, so that all dental surfaces can be easily reached. The bristles are constructed of a special polyene plastic (Rislan®) that has greater flexibility and a lower capacity for water absorption than nylon. The end of each bristle has been rounded to prevent mechanical damage to either dental enamel or gingival mucosae.[4, 10, 11] The brush head may be sterilized by boiling or autoclaving without damage. Since the brush heads are interchangeable, all members of a family can use one instrument, each having his own brush head.

The Patients. Twenty patients diagnosed as having periodontitis characterized by deposits of materia alba, redness of papillary and marginal gingivae, and lack of stippling of the gingivae participated in the present investigation. All of these patients were treated routinely by root planing. The entire series was divided into 2 groups of 10; one group was instructed in the use of the electrically operated instrument, while the 10 patients in the other group used conventional hand toothbrushes and served as controls. The experiment was conducted over a period of 6 weeks.

All of the patients returned weekly for further treatment and evaluation of home care. The presence of materia alba was

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*Broxodent is a registered Squibb trade-mark.

scored, arbitrarily, in ascending order of size of deposits, as +, ++, 05, +++; the color of the gingivae was scored as red, reddish pink, or pink, and stippling was scored as 0 (unstippled), + (stippled), or +− (evidence of stippling—Table I). The scores were converted into numbers for convenience in evaluation.* The mean numerical value of the scores was calculated weekly (Table II).

RESULTS

There was definite and measurable improvement in all 20 patients (Table I, Table II). In the group that used the electrically powered instrument, deposits of materia alba were reduced from +++ to + in every case, usually before the fourth week; the color of the gingivae was changed from red to pink in every case, usually by the fourth week; and the gingivae had become stippled in 9 of the 10 cases by the fourth week (Table I). In the 10 patients who used the conventional toothbrush, materia alba deposits decreased from +++ to + in most cases by the fifth week; the color of the gingivae was changed from red to pink in every case, usually by the fifth week, and the gingivae became stippled in 7 of the 10 patients by the fifth week (Table I). The composite scores for both groups are shown in Table II.

DISCUSSION

It is of considerable interest to note that there appears to be a definite correlation between the reduction of materia alba deposits and of redness of the gingivae with evidence of stippling. This correlation constitutes the well-established evidence for the importance of home care in the management of chronic periodontitis. While each patient was not seen every week, it is evident that there was definite improvement in the health of the gingivae in both groups at the end of 4 weeks (Table I,

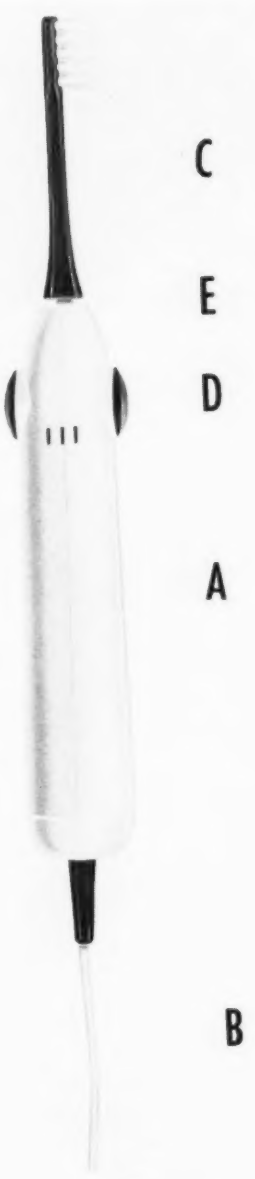

Fig. 1. The electrically operated toothbrush (Broxodent) used in the present study. Key: A, handle, containing electric motor in water-tight seal; B, power cord; C, replaceable brush head with slotted base; D, brake buttons; E, stud for attachment of brush head.

*The numerical values used in scoring were as follows: materia alba deposits, + = 1, + + = 2, + + + = 3; gingival coloring, red = 3, reddish pink = 2, pink = 1; gingival stippling, + − = 0.5, + = 1, + + = 2, + + + = 3.

TABLE I

Six-Week Clinical Record of Results of Home Care in 20 Periodontal Patients

Week No.	*Deposits of Material Alba**	*Color of Papillary and Marginal Gingivae*	*Gingival Stippling***
		Electrical Instrument	
	Patient No. 1		
1	+++	Red	0
2			
3	++	Reddish Pink	+
4			
5	+	Pink	++
6			
	Patient No. 2		
1	+++	Red	0
2	+++	Red	0
3			
4	++	Reddish Pink	+
5			
6	+	Pink	+++
	Patient No. 3		
1	+++	Red	0
2	+++	Reddish Pink	0
3			
4	++	Pink (−)	+
5			
6	+		
	Patient No. 4		
1	+++	Red	0
2	+	Reddish Pink	+−
3	+	Reddish Pink	+
4	+	Pink	+
5			
6			
	Patient No. 5		
1	+++	Red	0
2	+	Reddish Pink	+−
3	+	Reddish Pink	+
4	+	Pink	+
5			
6			

Week No.	*Deposits of Materia Alba**	*Color of Papillary and Marginal Gingivae*	*Gingival Stippling***
		Electrical Instrument	
	Patient No. 6		
1	+++	Red	0
2	++	Red	0
3	++	Reddish Pink	+−
4	++	Reddish Pink	+−
5			
6	+	Pink	+
	Patient No. 7		
1	+++	Red	0
2	++	Reddish Pink	+−
3	+	Pink (−)	+
4			
5			
6			
	Patient No. 8		
1	+++	Red	0
2			
3	+	Reddish Pink	+−
4	+	Pink	+
5			
6			
	Patient No. 9		
1	+++	Red	0
2	++	Reddish Pink	0
3			
4	+	Pink (−)	+
5	+	Pink	+
6			
	Patient No. 10		
1	+++	Red	0
2			
3	++	Reddish Pink	+−
4	+	Pink	+
5	+	Pink	+
6			

*Key: +++=large deposits, ++=moderate deposits, +=slight deposits.

**Key: 0=no stippling, +−=some stippling, +=well stippled.

TABLE I (continued)
Six-Week Clinical Record of Results of Home Care in 20 Periodontal Patients

Week No.	Deposits of Materia Alba*	Color of Papillary and Marginal Gingivae	Gingival Stippling**
	Conventional Toothbrush		
	Patient No. 1		
1	+++	Red	0
2			
3	++	Reddish Pink	+−
4			
5	+	Pink (−)	+
6	+	Pink	+
	Patient No. 2		
1			
2	++	Reddish Pink	+−
3	+	Pink	+
4	+	Pink	+
5			
6			
	Patient No. 3		
1	+++	Red	0
2	+++	Red	0
3	++	Reddish Pink	+−
4			
5	+	Pink	+
6			
	Patient No. 4		
1	+++	Red	0
2			
3	++	Reddish Pink	+−
4			
5	+	Pink	+
6			
	Patient No. 5		
1	+++	Red	0
2	++	Reddish Pink	+−
3			
4	+	Pink	+
5			
6			

Week No.	Deposits of Materia Alba*	Color of Papillary and Marginal Gingivae	Gingival Stippling**
	Conventional Toothbrush		
	Patient No. 6		
1	+++	Red	0
2	+++	Red	0
3	++	Reddish Pink	+−
4			
5			
6	+	Pink	−
	Patient No. 7		
1	+++	Red	0
2	++	Reddish Pink (+)	0
3	++	Reddish Pink (−)	+−
4	+	Reddish Pink	+
5	+	Pink	+
6	+	Pink	+
	Patient No. 8		
1	+++	Red	0
2	++	Reddish Pink	+−
3			
4	+	Pink	+
5			
6			
	Patient No. 9		
1	+++	Red	0
2	++	Reddish Pink	+−
3	++	Reddish Pink	+−
4	+	Pink	+
5			
6	+	Pink	+
	Patient No. 10		
1	+++	Red	0
2			
3	++	Reddish Pink	+−
4			
5	+	Pink	+
6			

*Key: +++ = large deposits, ++ = moderate deposits, + = slight deposits.
**Key: 0 = no stippling, +− = some stippling, + = well stippled.

TABLE II

Mean Weekly Scores of Clinical Performance in Home Care of 20 Periodontal Patients

Week No.	*Electrical Instrument*	*Conventional Toothbrush*
	Deposits of Materia Alba	
1	3.0	2.7
2	1.9	2.3
3	1.4	1.9
4	1.2	1.0
5	1.0	1.0
6	1.0	1.0
	Gingival Coloring	
1	3.0	3.0
2	2.3	2.3
3	2.0	1.8
4	1.1	1.2
5	1.0	1.0
6	1.0	1.0
	Gingival Stippling	
1	0	0
2	0.21	0.28
3	0.78	0.56
4	0.81	0.91
5	1.32	1.20
6	2.00	1.00

Table II). In fact, in every one of the 20 patients who participated in the present study, there was a change in the gingival coloring from red to reddish pink or pink and stippling, or at least evidence of stippling. After 4 weeks there appears to be rapid resolution to pink, stippled gingivae, with only minimal evidence of materia alba.

There appeared to be nearly identical improvement in all 20 patients, whether they used the manual toothbrush or the electrically powered instrument. It is noteworthy, however, that continued improvement after the fourth week, characterized by stippling of the gingivae, appeared to proceed more rapidly in the patients who used the electrically powered device. The mean stippling at the end of 4 weeks was essentially the same in both groups, but at 6 weeks, when the trial was terminated, the gingival stippling in the patients who used the electrically powered device was greater than in the control group. This finding suggests that the rapid vertical oscillation of the brush head strokes the gingivae at much increased frequency over that of hand brushing and that greater stippling results. If employed for a given period of time, it is obvious that the electrically driven brush must massage the gingivae more effectively than is possible with a hand brush, due to the increased oscillating contact of the brush head with the gingivae.

SUMMARY

An electrically powered toothbrush (Broxodent) was used in the home care of 10 periodontal patients for a period of 6 weeks. An equal number of patients with the same condition used conventional toothbrushes during the same period and served as controls. All 20 patients were treated routinely by root planing and were seen weekly. The therapeutic effectiveness of the home treatment was evaluated at each visit according to relative freedom from deposits of materia alba and improvement in gingival color and stippling.

Great improvement was noted in all 20 patients, thus reaffirming the established correlation between decrease in deposits of materia alba and increase in gingival stippling. The results obtained in the group that used the electrically operated instrument and the control group were parallel, except that after the fourth week the improvement in gingival stippling was more marked in the group that used the electrical instrument. This result would appear to be related directly to the greater effectiveness of the electrically powered instrument in gingival massage.

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Wire Ligature, a Feasible Form of Temporary Splinting

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THE usual reason for splinting the anterior teeth is because of their loosening or splaying outward.[1] Under either circumstance, a period of immobilization is desirable, as pointed out by Dr. Bernard B. Saturen's paper[2] in its first paragraph with all its footnotes.[3,4,5,6,7] and his own last paragraph.[8]

Criticism[9] has been expressed by Dr. Saturen of the use of wire ligatures for splinting purposes. He has pointed out that the form of the anterior teeth of both jaws is conical, and that, thus, because of the components of force, there will result protrusion and possibly extrusion. While Dr. Saturen has justly emphasized the conical factors and the principle of component forces in physics, he has failed to take advantage of the other vectors of force in the mouth.[10] These factors can be utilized in wiring of the teeth.

Utilizing these other vectors of force, the conical components then will have a favorable reaction if a method, arbitrarily called "contact point wiring" to distinguish it from the usual wiring that lies in the middle or gingival thirds of the crowns, is employed. To do contact point wiring requires but a few moments, as compared to the time necessary for anchor wiring[11] and the like.

28 ga. stainless steel wire is used as the archwire, and a generous amount is taken for the archwire loop, so that there will be ample for bringing the arch from the gingival to the incisal regions. If bicuspids are present, they should be utilized as the anchor teeth, due to the conical factors of the cuspids. The archwire is placed around

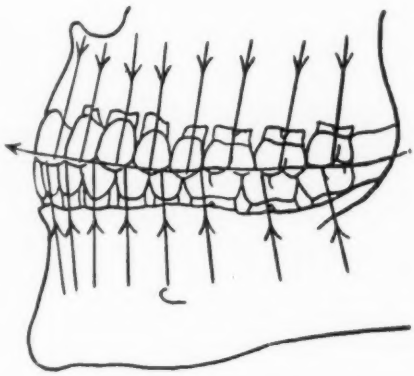

Fig. 1. Vectors of force (Salzmann).

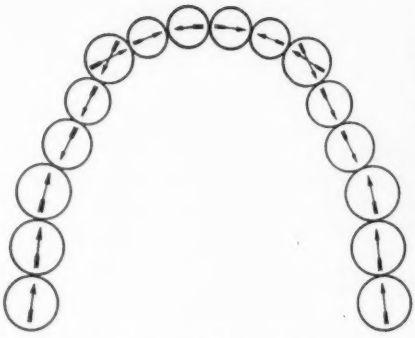

Fig. 2. Vectors of force (Salzmann).

Fig. 3. The usual form of anchor wiring which Dr. Saturnen justly criticized.

the neck of the bicuspid and an interproximal loop lightly twisted between bicuspid and cuspid at the round end of the archwire loop, which arbitrarily will be called the A-end to distinguish it from the B-end, or the twisted-wire end of the archwire. The archwire loop is brought up on both sides diagonally across both cuspids from distal to mesial, and at the contact point

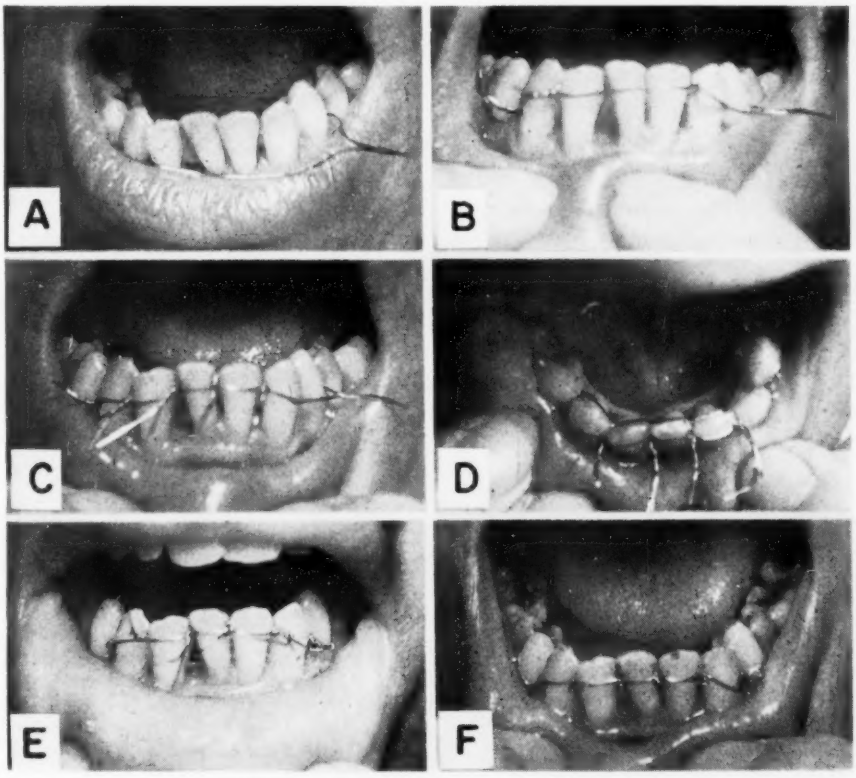

Fig. 4. (a) Initial form of loop. (b) Arch wire around cuspid and interproximals of laterals and cuspids. (c) Teeth apposition. (d) Lingual view of ligation. (e) Teeth apposition. (f) Incisal view with tracing paper, wire ends "tucked in."

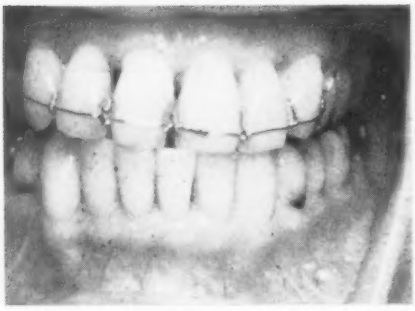

Fig. 5. Appearance of upper mouth ligation.

with each lateral, another interproximal loop is made. The A-end can be tightened, but the B-end should be left loosely twisted. The next interproximal loop made should be between the contact points of the centrals, and the ends again loosely twisted. The A-end of the lateral-central contact point then has its loop placed around the arch wires, and the ends tightened, then the B-end lateral-central receives the same treatment. Meanwhile, the fingers have been manipulating and aligning, if the teeth were loosened, the incisal arc, and when the teeth seem to be at their optimum, all contact point loops are tightened as is the B-end of the arch wire. The ends of the wires are cut and bent into the interproximals to reduce annoyance to the mucosa of the lips, the bite checked and ground if necessary. Apparently, this method of wiring was originated by Dr. Lillian Barkann,[12] has been forgotten, and rediscovered from time to time.

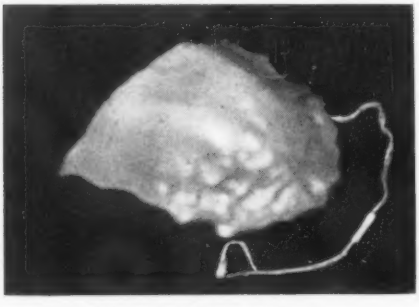

Fig. 6. Hawley retainer.

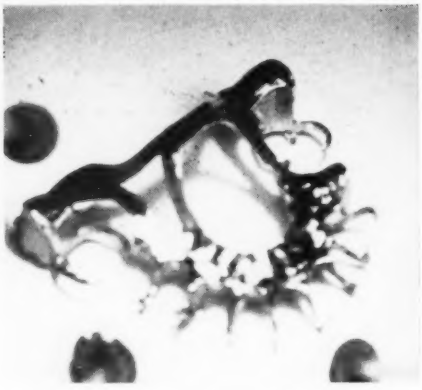

Fig. 7. Removable Finger Attachment.

Cleanliness is attained by the usual brushing manner, and appearance is not too conspicuous, as there is a modicum of wire displayed. Incidental attention usually consists of a tightening of the contact point loops, as the anterior vectors of force[13] regain control. There is seldom any breakage of these contact point loops, as contrasted to Dr. Saturen's statement.[14]

Retention of the anteriors should preferably be for a period of six months.[15] The lower teeth probably will have sufficient stability to do without further splinting by that time. The uppers, however, due to the tendency of the lowers to pound them outward, may have to have either a Hawley Splint for night wear, or a removable finger-attachment for a long period of wear. This latter can be utilized in removable bridgework should the patient have lost posterior teeth.

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Evaluation of a Desensitizing Agent By Response to a Controlled External Stimulus

by LOUIS R. BURMAN, A.M., D.D.S.,* AND ABRAHAM GOLDSTEIN, D.D.S.,**
New York, N. Y.

THE sensitive tooth has long been, and remains, a constant source of annoyance to both the patient and the dentist. As far back as one can trace the dental literature, there has been a constant search for improved agents to cope with this problem.

Various authors have suggested that dentine encroachment due to loss of enamel or cementum,[1,2,3] no matter what the etiology, tends towards tooth sensitivity. It has been stated that dentinal fibrils connecting the odontoblasts to the pulpal tissue form the pathway for transmission of sensations from the external environment.[4,5,6,7]

There have been many chemicals, and even mechanical devices,[8] proposed and used for the purpose of desensitizing teeth. These include silver nitrate solution, zinc chloride solution,[9] sodium fluoride in paste or liquid form,[9,10] paraformaldehyde powder,[11] formalin,[12] Andresen's powder,[13] hot olive oil,[14] and Gottlieb's solutions.[15] These agents have produced varying degrees of tooth desensitization. This variability in the results of therapy may be attributed to the fact that tooth sensitivity stems from a number of different causes, and reduction of sensitivity to one cause does not necessarily eliminate sensitivity due to other etiologic factors.[1] Teeth may be sensitive to hot or cold foods, air, temperature[16,17] and pressure changes, trauma, sweets, acids,[17] and such mechanical stimuli as instrumentation.[2,15]

Fitzgerald in 1956 reported excellent desensitization results in most of a series of 92 patients with the use of 'Thermodent' Tooth Paste. His results were based on the subjective responses of the patients.[18]

In 1958, in a series of 74 patients, Abel reported a marked benefit in the reduction of sensitivity with the same tooth paste. Here, again, the author relied on the patients' subjective symptoms.[19]

Toto, Staffileno and Gargiulo in 1958 tested this tooth paste and a placebo in 71 patients undergoing periodontal treatment. They reported beneficial results, again rely-

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Materials used in this study were supplied by Thos. Leeming & Co., Inc., New York, N.Y.

TABLE I
Location of Hypersensitive Teeth

Upper right molars	4	Lower right molars	8
Upper right bicuspids	14	Lower right bicuspids	12
Upper left molars	2	Lower left molars	5
Upper left bicuspids	7	Lower left bicuspids	14
Upper incisors and cuspids	28	Lower incisors and cuspids	29

Total molars involved	19	
Total bicuspids involved	47	
Total incisors and cuspids involved	57	
	123	
Labial and buccal cervical surfaces involved		112
Lingual and palatal cervical surfaces involved		3
Interproximal surfaces involved		3
Incisal or morsal surfaces involved		5
		123

ing on the patients' own subjective responses.[20]

In our attempt to evaluate the effectiveness of a desensitizing agent, it was thought advisable to choose a method of testing that would reduce the possible errors caused by the patients' subjective observations. A pulp testing apparatus was tried since it was hoped that definite degrees of sensitivity might be registered for a tooth surface in a purely objective manner. It was found that the electrode used was too large for minute areas. The electrode was reduced in size but this did not allow for retention of liquid needed for effective use of the instrument. It was also determined that the moisture necessary to make the electric contact was uncontrollable and therefore interfered with proper registration of the current.

In a preliminary examination of many patients, it was found that the best objective results were obtained with a sharp explorer as it travelled a course over the tooth surface. Using a similar pressure on all patients, there was a definite response to the instrument's touch when a sensitive area was encountered, and no reaction at all when there was no difficulty with the tooth tested. In this manner, it was possible to chart the exact location of tooth sensitivity without regard to the patient's subjective comments about his sensitivity. It should be emphasized that these tests in this series of patients show results only in relation to the controlled touch of the explorer. Other subjective remarks made by the patients were recorded, but are not reported except in a later general statement, and are not related to our results.

METHOD

Patients were examined at random using a number 23 sharp explorer. The instrument was passed along the circumference of the tooth with special regard for the cervical portion. Incisal, morsal and interproximal surfaces were also tested. Only those patients giving a definite and positive response were used. The involved tooth was indicated on a chart together with the

TABLE II
Results of Treatment

		No.	*%*
Very good result	(++)	91	74
Good result	(+)	21	17
Questionable benefit	(±)	6	5
No benefit	(−)	5	4
		123	100%

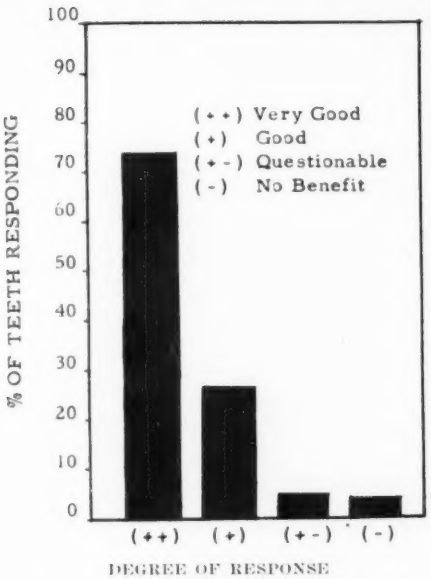

Fig. 1. Response of 123 hypersensitive teeth to 'Thermodent' Tooth Paste as objectively measured by reaction to a controlled stimulus.

exact area of sensitivity. No attempt was made to determine the etiology of the sensitivity. Oral hygiene, brushing habits, dietary habits, etc., were not used as a criteria for patient acceptance for testing. The only qualifying requirement was that the area in question had been sensitive for some time. Carious portions of teeth were not used in this experiment. Restorative and periodontal dental care was not stopped and the patient was maintained in his normal dental routine.

Each patient was supplied with a multi-tufted nylon bristle brush, soft in texture, and a tube of tooth paste bearing a code number. The actual content of the tube was a dentifrice containing precipitated chalk, salts, a flavoring agent and 1.4% formalin.* The patient was instructed to place some of the tooth paste on the brush and to burnish it into the area of sensitivity. This was to be done twice a day, morning and night, in addition to the routine brushing[17] done by the patient for the rest of the mouth.

Checking of the sensitive areas was done after a two week period.

*"Thermodent" Tooth Paste.

RESULTS

Tabulation was made in response to the touch of the explorer with the following notations:

++very good result
+good result
±questionable benefit
−no benefit

The patient's subjective responses were noted. While the age and sex of the patients were recorded, no correlation was attempted between these categories and the degree of sensitivity or the results of the use of the tooth paste.

One hundred and twenty-three individual teeth were tested in fifty-nine patients and, in addition, three patients were tested subjectively for full mouth generalized sensitivity. Six other patients were included in the testing program but final results were not obtained.

Twenty-one males (average age 31) and 48 females (average age 41) were included in this series. The location of the hypersensitive areas found are reported in Table I as being of possible interest in future studies of hypersensitivity.

Following the method outlined above, and using as the criterion of evaluation the response to a controlled external stimulus, 112 (91%) of the 123 sensitive teeth under observation were benefitted by the use of 'Thermodent' (Table 2) and (Fig. 1).

CONCLUSIONS

The results of our testing program on the response of individual teeth to controlled touch sensitivity showed an extremely high percentage of beneficial results after two weeks of treatment. The three whole mouth cases treated reported subjectively that there was generalized re-

duction of sensitivity. Since 91% of the teeth treated with 'Thermodent' Tooth Paste for reduction of touch sensitivity benefited as a result of the treatment, this desensitizing agent appears to be valuable in the reduction of this type of sensitivity.

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Bernard M. Walker, D.D.S.

Charlotte, North Carolina

We have just been informed that Dr. Bernard M. Walker died in Charlotte, North Carolina, at the age of 51. Dr. Walker was very active in the Southern Academy of Periodontology and the American Academy of Periodontology. He was also a diplomate of the American Board of Periodontology.

Dimensions and Relations of the Dentogingival Junction in Humans

by ANTHONY W. GARGIULO, D.D.S., M.S., FRANK M. WENTZ, D.D.S., PH.D., AND BALINT ORBAN, M.D., D.D.S.*

IN 1921, Gottlieb's discovery of the epithelial attachment of the gingiva opened new horizons which served as the basis for a better understanding of the biology of the dental supporting tissues in health and disease. Three years later his pupils, Orban and Kohler (1924), undertook the task of measuring the epithelial attachment as well as the surrounding tissue relations during the four phases of passive eruption of the tooth. Gottlieb and Orban's descriptions of the epithelial attachment unveiled the exact morphology of this epithelial structure, and clarified the relation of this structure to the enamel of the tooth.

In recent years the prevailing concept of the epithelial attachment was challenged by Waerbaug.[3] He returned to the old concept of a potential space extending from the gingival margin to the cementoenamel junction. Waerhaug's altered convictions were based upon several observations. These are: (1) he was able to insert a thin steel blade into this space without pressure. From histologic sections, he claimed that there was no difference between the epithelium of the intact areas and in the area where the blade was inserted. (2) In addition he claimed that after a gingival flap had been pulled away from the enamel surface and the flap repositioned, no difference could be seen between the operated and the non-operated areas. Repetition of these procedures by Orban[4] have shown Waerhaug's findings could not be verified under similar experiments. Gottlieb's discovery was in the least reconfirmed; however Waerhaug's challenge was not without benefit. The so-called strength of adherence of the epithelial attachment, and the organic nature of the attachment had to be reconsidered. The author's are now inclined to subscribe to Weski's[5] idea; mainly that the epithelium after enamel maturation, produces a cementing substance that attaches the epithelium to the enamel surface and later to the surface of the cementum. It now seems impossible that Gottlieb's original idea of a union between ameloblast and the forming and maturing enamel rods could survive the final calcification of the enamel matrix.

Discussions with Sicher (1959)[6] lead to the reconsideration of the mode of attachment and the formulation of a physiologic division of labor of the supporting tissues at the "dento-gingival junction." This established the concept of the dentogingival junction as a functional unit composed of two parts: (1) the connective tissue fibrous attachment of the gingiva and (2) the epithelial attachment. The two separate components share a division of function.

The biologic protection of the dentogingival junction is the function of the epithelial attachment. The epithelium attaches to the circumference of the tooth as a broad band the "attached epithelial cuff." The epithelial attachment to the tooth is not firmly attached in spite of the fact that it is stronger than the individual cohesiveness of the epithelial cells. The firmness of the gingival attachment to the tooth is derived by the fibrous connective tissue bound to the cementum, alveolar bone and gingiva.

Because of the dynamic alterations in the component parts of the dentogingival junction it is important to know their positions in all phases of eruption under normal conditions. The importance of this relation is enhanced when one considers the imbalance of these components in periodontal disease. Thus, these dimensions can serve as a base line for future studies involving the pathologic status of the dentogingival junction and serve as "the physiologic dentogingival junction."

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*Dr. Orban died June 1, 1960.

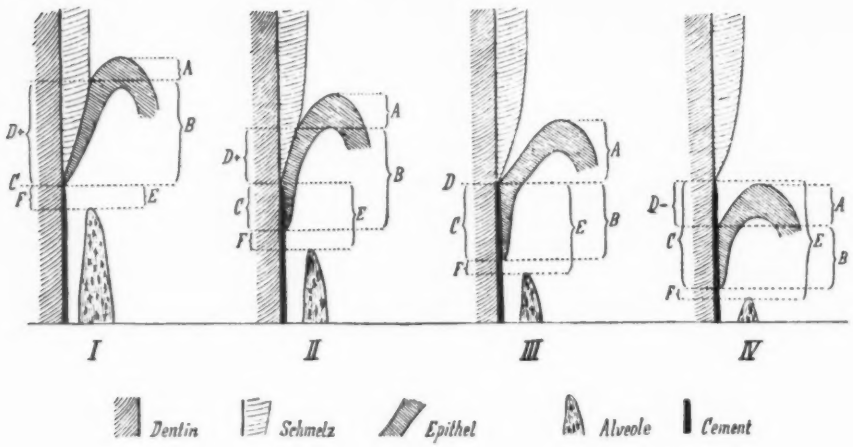

Figure No. 1

The present study consisted of two parts; first to re-evaluate the measurements in the Orban-Kohler paper, but now as part of the dentogingival junction and not as a single isolated structure; and second to add new measurements. It also establishes a norm for the dentogingival junction in all phases, chronologic ages, surfaces and six measured distances.

MATERIAL AND METHOD

The material for this study were the measurements obtained from human autopsy specimens by Dr. Balint Orban in earlier studies. Additional measurements were obtained from 30 human jaws in the collection of Dr. Rudolph Kronfeld. All specimens were taken at autopsy as block sections in order to obtain the component parts of the dentogingival junction intact. The age range was from 19 to 50 years. The specimens were fixed in 10% formalin, embedded in cellodin and 15-20u sections cut. The sections were stained in hemotoxylin and eosin.

The measurements were made from the microscopic specimens of 30 jaws, 287 individual teeth and their respective dentogingival components. A total of 325 surfaces were measured; of these 83 were on the mesial surface, 82 were on the distal surface, 82 on the vestibular surface and 78 on the oral surface. All specimens were free of extensive pathology and fulfilled the requirements of clinically normal specimens. The actual measurements were made with a disc micrometer, and all are recorded in millimeters.

A total of six different measurements were made for each individual specimen. These areas changed their relations in the four different phases of eruption in which measurements were made. In Figure 1, one can observe the various phases of passive exposure as established by Orban and Kohler in 1934.

The areas measured were: a) depth of the gingival sulcus, b) length of the attached epithelium, c) most apical point of the epithelial attachment from the cemento-enamel junction, d) distance from the base of the sulcus to the cemento-enamel junction, e) distance of the cemento-enamel junction from the alveolar bone, f) distance from the most apical point of the epithelial attachment to the alveolar bone (connective tissue).

FINDINGS

The findings from each of the phases were analyzed according to the six individual measurements. All surfaces (mesial,

TABLE I
Phase I Analysis

Measurement	*Range (mm)*	*Mean Average (mm)*
A. Sulcus depth	0.00 to 2.62	.80
B. Attached epithelium	0.28 to 3.72	1.35
C. Apical point of Epithelial Attachment below Cemento-Enamel Junction	0.00 to 0.00	0.00
D. Bottom of Sulcus from Cemento Enamel Junction	+0.28 to 3.36	+1.35
E. Cemento Enamel Junction to Alveolar Bone	0.04 to 3.36	1.08
F. Deepest point of Epithelial Attachment to Alveolar Bone	0.04 to 3.36	1.08

distal, vestibular, oral) were placed in one average value for the given measurement. The analysis first was done according to each of the individual phases of eruption in the dynamic relation existing at the dentogingival junction throughout life. Results of the measurements were as shown in the following tables.

The number of specimen surfaces in the phase I analysis were 25. The total length of the dentogingival unit, (from the tip of the gingival margin to the crest of the alveolar bone) A+B+F, represents the magnitude of the dentogingival junction. For phase I this magnitude was 3.23 mm. The total attachment (B+F) was 2.43 mm. The average age for phase I was 24.5 years and the specimens ranged from 19 to 43 years.

Due to the definite anatomy of phase I, measurement C was zero, those for B, and D; and E, and F, were equal. However, this relation is altered as the epithelial attachment progresses in an apical direction in the later phases.

One hundred and twenty-six (126) specimen surfaces were analyzed in phase II. The total length of the dentogingival unit (A+B+F+) was 3.06 mm. The total attachment (B+F) was 2.45 mm. The average age for phase II was 31.4 years, and ranged from 19 to 50 years.

Phase III is a highly precise arrangement of the dentogingival apparatus, and the least number of specimens were found at this phase. In phase III eleven specimen surfaces were measured.

There was no recorded reading for "D" since in the phase III relation, the base of the sulcus is just at the cemento-enamel junction.

TABLE II
Phase II Analysis

Measurement	*Range (mm)*	*Mean Average (mm)*
A. Sulcus depth	0.00 to 5.36	.61
B. Attached epithelium	0.34 to 2.90	1.10
C. Apical point of Epithelial Attachment below Cemento-Enamel Junction	0.03 to 2.36	0.43
D. Bottom of Sulcus from Cemento Enamel Junction	+0.02 to 2.60	+0.68
E. Cemento Enamel Junction to Alveolar Bone	0.35 to 5.00	1.55
F. Deepest point of Epithelial Attachment to Alveolar Bone	0.02 to 4.38	1.07

TABLE III
Phase III Analysis

Measurement	Range (mm)	Mean Average (mm)
A. Sulcus depth	0.00 to 0.94	.61
B. Attached epithelium	0.16 to 1.04	.74
C. Apical point of Epithelial Attachment below Cemento-Enamel Junction	0.16 to 1.04	.74
D. Bottom of Sulcus from Cemento Enamel Junction	0.00 to 0.00	0.00
E. Cemento Enamel Junction to Alveolar Bone	0.88 to 3.20	1.71
F. Deepest point of Epithelial Attachment to Alveolar Bone	0.16 to 2.37	1.05

The total length of the dentogingival unit (A+B+F) was 2.41 mm. The total attachment (B+F) was 1.80 mm. The average age for phase III was 32.3 years, and ranged from 22 to 50 years.

One hundred and sixty-three (163) specimen surfaces were measured in phase IV. The total length of the dentogingival unit (A+B+F) was 2.53 mm. The total attachment (B+F) was 1.77 mm. The average age for phase IV was 39.7 years, and ranged from 20 to 50 years.

The previous analysis was a total average of all surfaces. In order to obtain the total mean average all tooth surfaces were measured for each of the distances, thus one can calculate the mean average for each of the 4 tooth surfaces. From this one can see the variance which occurs on the four different surfaces of the same tooth and for the same area measured. See Tables V to X.

In an earlier paper by Stanley[7] he states that "the type of tooth and type of tooth surface (mesial or distal) has no effect on the mean lengths of the distances." As can obviously be seen from the present study, the tooth surface is rather variable in the total circumference, and on each of the individual teeth.

DISCUSSION

This work has indicated that there is a somewhat definite proportional dimensional relation between the dentogingival junction and the other supporting tissues of the tooth.

The validity of the concept of the dentogingival junction has been fortified, and the duality of its components (epithelial and fibrous connective tissue) has been identified as an orderly one.

One can no longer speak of passive exposure only being associated with the apical

TABLE IV
Phase IV Analysis

Measurement	Range (mm)	Mean Average (mm)
A. Sulcus depth	0.00 to 2.25	1.76
B. Attached epithelium	0.08 to 2.65	0.71
C. Apical point of Epithelium Attached below Cemento-Enamel Junction	0.39 to 6.08	1.41
D. Bottom of Sulcus from Cemento Enamel Junction	−0.03 to 5.84	−1.14
E. Cemento Enamel Junction to Alveolar Bone	1.10 to 10.88	2.81
F. Deepest point of Epithlial Attachment to Alveolar Bone	0.00 to 6.52	1.06

TABLE V
Measurement A.—Sulcus Depth

Phase	*Mesial*	*Distal*	*Vestibular*	*Oral*	*Total Average*
I	1.09	.84	.87	.40	.80
II	.51	.64	.76	.53	.61
III	.94	.50	.62	.41	.61
IV	.81	.87	.82	.57	.76

migration of the epithelial attachment. The correct interpretation of the gingiva and its relation to the tooth with increasing age can only be understood, if the connective tissue attachment is also considered. (See Table XI).

The physiologic apical shift of the dentogingival junction from stage to stage during passive eruption, is responsible for the passive exposure of the tooth and is not merely due to the "peeling back" of the epithelial attachment.

From chart XI, one can readily see that the epithelial attachment is our most variable, while the connective tissue attachment is the most constant measurement. This observation further supports Stanley's[7] finding that the "epithelial attachment appeared to be the most variable anatomic structure within the periodontium." Thus, during passive eruption the magnitude of the epithelial attachment diminishes. Early in phase I the amount of epithelial attachment is 1.35 mm. and decreases in phase IV to 0.71 mm., this represents a significant diminution. In correlating the epithelial attachment with age it was seen that there was less attachment with an increase in dental and physiologic age. *On the other hand the connective tissue component appears to be a constant through the stages of passive eruption.*

TABLE VI
Measurement B.—Length of Attached Epithelium

Phase	*Mesial*	*Distal*	*Vestibular*	*Oral*	*Total Average*
I	1.56	1.37	1.35	1.14	1.35
II	1.35	1.20	1.08	.80	1.10
III	.44	.88	.87	.77	.74
IV	.83	.88	.63	.53	.71

Upon closer interpretation the meanings of these data indicates a need for a revision of the phases of passive eruption. A further alignment of measurements, it is seen that the four phases of eruption can rightfully be classified into two divisions. The basis for this statement is further emphasized from Table XI. It is seen that the attached epithelium is significantly greater in phase I and II, than the attached epithelium is in phase III and IV. The significant thing is that the total attachment (B+F) measures similar in phase I and II (Division A); and phase III and IV (Division B). Therefore it is thought that we truly have only two divisions of passive eruption.

TABLE VII
Measurement C.—Distance of Attached Epithelium Below Cemento Enamel Junction

Phase	*Mesial*	*Distal*	*Vestibular*	*Oral*	*Total Average*
I	.00	.00	.00	.00	.00
II	.51	.45	.40	.36	.43
III	.44	.88	.87	.77	.74
IV	1.76	1.10	1.68	1.11	1.41

In addition it can be seen the greater amount of attached epithelium in phase I and II, and the greater amount of connective tissue attachment in phase III and IV. From this one can also see that in division A (phase I and II) the total attachment is of a greater magnitude than that of division B (phases III and IV).

In analyzing each of the measurements for the four phases we can make some rather revealing observations. In measurement A, sulcus depth, note that under circumstances of normal physiology this remains rather constant. For measurement B, the length of the attached epithelium, note that as one progresses from phase I to IV there is less attachment of epithelium to the tooth surface. From these findings one can see that there has been approximately

TABLE VIII

Measurement D.—Distance of Bottom of Sulcus from The Cemento Enamel Junction

Phase	Mesial	Distal	Vestibular	Oral	Total Average
I	1.56	1.37	1.35	1.14	+1.35
II	.88	.74	.68	.45	+.68
III	.00	.00	.00	.00	.00
IV	.93	1.14	1.06	1.45	−1.14

TABLE IX

Measurement E.—Distance of Cemento-Enamel Junction from Alveolar Bone

Phase	Mesial	Distal	Vestibular	Oral	Total Average
I	.75	1.10	1.01	1.49	1.08
II	1.39	1.42	1.86	1.56	1.55
III	1.46	1.58	2.40	1.42	1.71
IV	2.66	3.08	2.82	3.10	2.81

a 50% loss in magnitude from phase I to (1.33 mm.) to phase IV (.71 mm.). In measurement C., the most apical point of the epithelial attachment below the cemento-enamel junction, there is an increase in the clinical crown which is a normal course from phase I to IV as the epithelial attachments shifts in an apical direction. Measurement D, the distance of the bottom of the sulcus from the cemento-enamel junction, is another shift in an apical direction with a progression from phase I to IV.

Measurement E, distance of the cemento-enamel junction from the alveolar bone, is a value which increases from phase I to IV. This is interesting in that as the tooth undergoes active eruption the alveolar bone crest doesn't appear to keep "pace" with the cemento-enamel junction. However, this no doubt is due to the passive exposure of the tooth at the dentogingival junction which is occurring simultaneously with active eruption. Measurement F, the distance from the base of the epithelial attachment to the alveolar bone, represents the connective tissue attachment of the gingival fibers and appears to be the most constant value that the normal dentogingival junction possesses. The values were so close that 3 of the 4 phases almost duplicate each other to the hundredth of a millimeter.

TABLE X

Measurement F.—Distance from Base of Attached Epithelium to Aveolar Bone (Connective Tissue Attachment)

Phase	Mesial	Distal	Vestibular	Oral	Total Average
I	.75	1.10	1.01	1.49	1.08
II	.81	.96	1.21	1.32	1.07
III	1.02	.69	1.53	1.03	1.06
IV	.89	1.09	1.22	1.07	1.06

TABLE XI

Dentogingival Junction
Total Attachment
(Measurement B+F)

	Phase and Environment	Total Attachment (mm) Length of Epithelial Attachment—B	Connective Tissue Attachment—F	B+F
	I Attachment on Enamel	1.35	1.08	2.43
DIVISION A				
	II Attachment on Enamel and Cementum	1.10	1.07	2.17
	III Attachment on Cementum	0.74	1.05	1.80
DIVISION B				
	IV Attachment on Cementum	0.71	1.06	1.77

TABLE XII
DENTOGINGIVAL JUNCTION
Total Average Magnitude
Sulcus Depth (A)
Epithelial Attachment (B) and
Connective Tissue Attachment (F)

	Average Measurement in mm.
Sulcus Depth (A)	.69 mm.
Length of Epithelial Attachment (B)	.97 mm.
Connective Tissue Attachment (F)	1.07 mm.

The total magnitude, or length of the dentogingival junction is seen to decrease from phase I to IV. From this observation we can see an apparent loss of total attachment to the tooth enamel and cementum. This is evidenced by the fact that the magnitude in phase 1 is 3.23 mm. and regresses to 2.53 mm. in phase IV.

While the preceding findings are of great value in themselves, one must fully realize the impetus this has upon pathology of the dentogingival junction and what the effect of surgical encroachment upon these areas has on the final dimensional proportions. This study will serve as a basis by which to understand the results obtained in osseous surgery and other approaches which denude and expose the dentogingival junction.

In an analysis of age, these findings would support the fact that about any chronologic age can be found in any of the four phases. Usually phase III and IV were absent in the chronologic ages below 21 years. In addition the average age here reported can create a false impression since only a given age range of 19 to 50 years was used.

SUMMARY

The prevailing concepts of the dentogingival junction are reviewed to re-emphasize the importance of this functional unit with its two component parts, 1) The connective tissue fibrous attachment and, 2) the epithelial attachment.

The dimensions of the dentogingival junction in four phases of passive eruption were ascertained from earlier studies of Orban and from thirty additional human jaws. A total of six different measurements were made on all types and surfaces of teeth. Upon comparison of all measurements we can observe that the previous four phases of eruption can be categorized in two divisions.

Of the measurements made, the distance from the base of the epithelial attachment to the crest of the alveolar bone (connective tissue attachment), is the most constant. It had a mean average length in all phases of 1.07 mm. The most variable part of the dentogingival junction was the length of the epithelial attachment.

This study will permit a better understanding of the alterations which occur at the dentogingival junction following osseous surgery, and surgical exposure of the dentogingival junction.

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Announcements . . . .

ROWE SMITH MEMORIAL FOUNDATION EIGHTH ANNUAL SEMINAR

The Rowe Smith Memorial Foundation Eighth Annual Seminar will be held November 30, 1961 to December 3, 1961, at the Fly-n-Fish Lodge, Karnack, Texas. The subject will be "Disturbed Carbohydrate Metabolism"; topics to be covered include epidemiology of disturbed carbohydrate metabolism; biochemistry of disturbed carbohydrate metabolism at the cellular level; dietary and hormonal influences on blood sugar level; methods of detection of disturbances of carbohydrate metabolism; clinical types of diabetes mellitus; anthropometry and carbohydrate metabolism; hypoglycemia; the influence of emotional factors on glucose metabolism; the treatment of diabetes mellitus. The moderator will be D. Walter Cohen, D.D.S., Director of Periodontia, University of Pennsylvania School of Dentistry.

Essayists include Donald W. Seldin, B.A., M.D., Professor and Chairman, Department of Internal Medicine, University of Texas Southwestern Medical School, and E. Cheraskin, M.D., D.M.D., Chairman of the Department of Oral Medicine, University of Alabama Dental School. For further information write Rowe Smith Memorial Foundation, 501 State National Bank Building, Texarkana, Arkansas.

UNIVERSITY OF WASHINGTON

The Graduate Division of the University of Washington School of Dentistry announces a graduate course in endodontics and microbiology leading to a master's degree in dentistry. The training period is two calendar years, beginning September, 1961.

The trainee will be supported on a stipend of $5300 a year received from a National Institutes of Health Teacher Training Grant. His tuition and some additional expenses will be paid as well as allowances for travel to scientific meetings.

The program is under the direction of Dr. John I. Ingle and includes advanced instruction in the basic sciences as well as the clinical and hospital disciplines. An extensive research project and thesis are required.

Inquiries should be directed to: Dr. Saul Schluger, Director, Graduate Dental Education, University of Washington, School of Dentistry, Seattle 5, Washington.

Robert L. Dement, D.D.S.

Atlanta, Georgia

June 15, 1961

Dr. Robert Dement of Atlanta, Georgia, passed away on June 15, 1961. Further details will be published in the next issue of the Journal.